A PROSPECTIVE STUDY OF PRE-OPERATIVE PREDICTORS OF DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY

Dissertation submitted to

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With fulfillment of the regulations for the award of the degree of

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DEPARTMENT OF GENERAL SURGERY

MADURAI MEDICAL COLLEGE

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CERTIFICATE BY THE GUIDE AND THE HEAD OF THE DEPARTMENT

This is to certify that the dissertation entitled "A PROSPECTIVE STUDY OF PRE-OPERATIVE PREDICTORS OF DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY" submitted by Dr.P.THIYAGARAJAN to the Tamil Nadu Dr M.G.R. Medical University, Chennai in partial fulfillment of the requirement or the award of M.S Degree Branch - I (General Surgery) is a bonafide research work was carried out by her under direct supervision and guidance from September 2013 to August 2014 in the Department of General Surgery, Madurai Medical College.

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DECLARATION

I, **Dr. P.THIYAGARAJAN,** declare that, I carried out this work on, A PROSPECTIVE STUDY OF PRE-OPERATIVE PREDICTORS OF DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY at the Department of General Surgery, Madurai Medical College during the period of March 2013 to August 2014. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award,degree, diploma to any other University, Board either in India or abroad. This is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S. degree examination in General Surgery.

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LIST OF ABBREVIATIONS USED

CHD	Common Hepatic Duct
CBD	Common Bile Duct
LC	Laparoscopic Cholecystectomy
TPN	Total parentral nutrition
USG	Ultrasonography
RHA	Right Hepatic Artery
GB	Gall Bladder
ERCP	EndoscopicRetrogradeCholangiopancreatography
P/C	Pericholecysticcollection
Pvalue	Predictivevalue
SGOT	SerumGlutamicOxaloaceticTransaminase
SGPT	SerumGlutamicPyruvateTransaminase

ABSTRACT

BACKGROUND

Cholelithiasisis the most common biliary pathology, with a prevalence of 10 to 15%. It is symptomatic in approximately 1to2% of patients. N I H consensus development stated that laparoscopic cholecystectomy "Provides a Safe and Effective treatment for most patients with symptomatic gallstones". In about 5 to 10% of laparoscopic cholecystectomy, conversion to open cholecystectomy may be needed for safe removal of gall bladder.

OBJECTIVES

To determine the predictive factors for difficult laparoscopic cholecystectomy

To study the clinical presentation of cholelithiasis

METHODS

The material for the present study comprised of 50 cases admitted to Govt Rajaji hospital Madurai from March 2013 to august 2014 for a period of 18 months. The cases confirmed by Ultrasonography were evaluated with following risk factors: age > 50years, male sex, BMI 25.1 to 27.5 and >27.5, previous surgery, prior hospitalization, palpable gall bladder, gall bladder wall thickening, impacted stone,

pericholecystic fluid collection. Each risk factor was given a score. The total score upto 5 predicted easy, 6 to 10 difficult and more than 10 very difficult.

RESULTS

The highest age incidence of cholelithiasis was in the 4^{th} decade, and was more common in females. Pain abdomen was the most common symptom. Ultrasonography detected gall bladder stones in all patients, wall thickening in 15 and pericholecystic fluid collection in 10. BMI >27.5 ($P<0.001$), history of prior hospitalization ($P<0.0008$), palpable gallbladder ($p<0.0364$), impacted stone ($P<0.0103$) and Pericholecystic fluid collection ($P<0.0471$) were significant predictors of difficult laparoscopic cholecystectomy.

INTERPRETATION AND CONCLUSION

The proposed scoring system had a positive prediction value for easy prediction of 94.7% and for difficult prediction of 100%.The conversion rate from laparoscopic cholecystectomy to open cholecystectomy was 10%.

KEYWORDS: LAPAROSCOPIC CHOLECYSTECTOMY, PREDICTIVE FACTORS, SCORINGSYSTEM

CONTENTS **PAGE NO**

LIST OF TABLES **PAGE NO**

LIST OF FIGURES PAGE NO

INTRODUCTION

Cholelithiasis is the most common biliary pathology. Gallstones are present in 10 to 15% of the general population and asymptomatic in the majority (>80%).The prevalence of gallstone varies widely in different parts of the world. In India it is estimated to be around 4%.

An epidemiological study restricted to rail road workers showed that north Indians have 7 times higher occurrence of gallstones as compared to south Indians.

Changing incidence in India is mainly attributed to westernization and availability of investigation that is ultrasound in both rural and urban areas and due to change in socioeconomic structure.

Approximately 1-2% of asymptomatic patients will develop symptoms requiring cholecystectomy per year

Cholelithiasis is rare in the first two decades. Incidence gradually increases after 21 years and reaches its peak in 5^{th} and 6^{th} decade. Women are more affected than men in the ratio of 4:1.

In 1992, The National Institute of Health (NIH) consensus development conference stated that laparoscopic cholecystectomy "provides a safe and effective treatment for most patients with symptomatic gallstones."

The advantages of laparoscopic cholecystectomy over open cholecystectomy are quick recovery of bowel functions, minimal pain in postoperative period, informed cosmesis, minimal hospital stay, earlier return to normal activity, and decreased overall cost.

Laparoscopic cholecystectomy has become the gold standard in the treatment of gallbladder pathology and is replacing open cholecystectomy. The rate of conversion from laparoscopic cholecystectomy to open cholecystectomy is 5 to 10%. Hence it is necessary to study the predictive factors for difficult laparoscopic cholecystectomy. Therefore this study was undertaken.

OBJECTIVES OF THE STUDY

To determine the predictive factors for difficult laparoscopic cholecystectomy.

To study the clinical presentation of cholelithiasis.

REVIEW OF LITERATURE

HISTORICAL ASPECTS

The Roman Celsus in his text, De Medicina (translated by W.G. Spencer in 1935), mentioned the liver and described its anatomic location in an accurate form: “The liver, which starts from the actual partition under the precordia on the right side, is concave within (that is on the inferior surface) and convex without; its projecting part rests lightly on the stomach and it is divided into four lobes. Outside its lower part, the gallbladder adheres to it.”

Vesalius found (that he had) a hemoperitoneum coming from an abscess which had eroded the portal vein. The gallbladder was yellow and contained 18 calculi. Very light, of a triangular shape with even edges and surfaces everywhere, green by color somewhat blackish. The spleen was very large.”

Morgagni published in 1769 an analysis of disease under the title Seats and Causes of Disease, among which are those of the liver and biliary tract.

Vater (1684-1751) was the first to describe the papilla of the duodenum.

Pettit introduced the term biliary colic

1878: Kocher performed a cholecystostomy in two stages (Glenn, 1971). In the first stage, he packed the wound with gauze to the bottom of the gallbladder, and 8 days later he emptied the residual stones from the gallbladder.

1885: Tait performed first cholecystostomy for gallbladder lithiasis in one stage.

1882: Langenbuch performed first elective cholecystectomy

1882: Von Winiwarter developed Cholecystenterostomy.

1895: Kocher wrote an article on internal choledochoduodenostomy to remove supra-ampullary choledochal calculi.

1897: Kehr placed a rubber tube in the common bile duct through the cystic duct; this was the first systematic use of biliary intubation.

1898: Thornton performed the first removal of a stone from the common bile duct.

1898: MacBurney published his experience with duodenostomy and papillotomy in patients with impacted periampullary calculi.

1898: Buxbaum observed biliary calculi on plain x-rays.

1912: Kehr developed T-tube.

1923: Bakes developed choledochoscopy.

1924: Graham developed oral cholecystography.

1932: Mirizzi developed Postoperative cholangiography.

1937: Mirizzi developed Intraoperative cholangiography.

1989: Dubois in Paris published the first series of laparoscopic cholecystectomies (Dubois et al).

HISTORY OF LAPAROSCOPY AND LAPAROSCOPIC CHOLECYSTECTOMY:

Laparoscopy(from the Greek, Laparo meaning the flank and Skopein meaning to examine), was first performed in 1901 by George killing of Dresden, Germany using room air filtered through sterile cotton for pneumoperitoneum and a wide

cystoscope to veiw the abdominal cavity of dog.The use of carbon dioxide (co2) for pneumoperitoneum was first recommended by Richard Zollikofer of Switzerland in 1924.

The primary mode of insufflation was the Veress needle which was introduced by Janos Veress of Hungary in 1938.

In 1933, A German general surgeon, Feowers, was the first to report laparoscopic lysis of abdominal adhesions for the diagnosis of bowel obstructions.

Kurt Semm incorporated new aspects of fiber optic and used automatic gas insufflator which allowed precise controlled intra abdominal pressure.

In 1983, Lukichev and colleagues described laparoscopic cholecystectomy for acute cholecystitis.

In 1985, Muhe of Boblinger, Germany performed the first laparoscopic assisted cholecystectomy.

In 1987,a French surgeon in Lyon, Phillipe Mouret, performed the first video-laparoscopic cholecystectomy.

ANATOMY

The extra-hepatic biliary tree consists of the right and left hepatic ducts, common hepatic duct, cystic duct and gallbladder and the common bile duct.

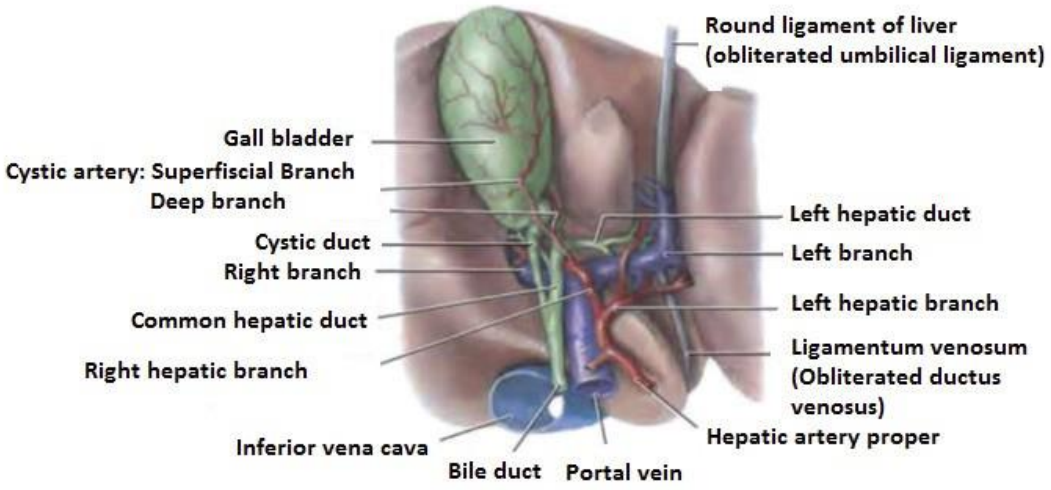


FIGURE1: showing anatomy of gall bladder, inferior view

GALL BLADDER:

The gall bladder is a flask-shaped, blind-ending diverticulum attached to the common bile duct by the cystic duct. It usually lies in a shallow fossa in the liver parenchyma covered by peritoneum continued from the liver surface. This attachment can vary widely. The gall bladder lies on a fibrous or cystic plate, which is part of

the perihilar system of fibrous tissue. The cystic plate attaches directly onto the anterior surface of the right portal pedicle. The hepatic parenchyma lies deep to the cystic plate, through which small bile ducts may penetrate to enter the gallbladder.Between the muscularis of the gallbladder and the cystic plate, a thin layer of areolar tissue thickens progressively from the top of the gallbladder downward. During dissection of the gallbladder from the liver, the posterior surface of the cystic artery and bile duct will be reached when the areolar tissue is left on the cystic plate. Should dissection be undertaken deep into the cystic plate, the surface to the right portal pedicle may be breached and result in injury to the right portal pedicle structures and the right hepatic duct

NECK:

Neck lies at the medial end close to the porta hepatis, and almost always has a short peritoneal cover attached to the liver (MESENTERY); this mesentery usually contains the cystic artery.

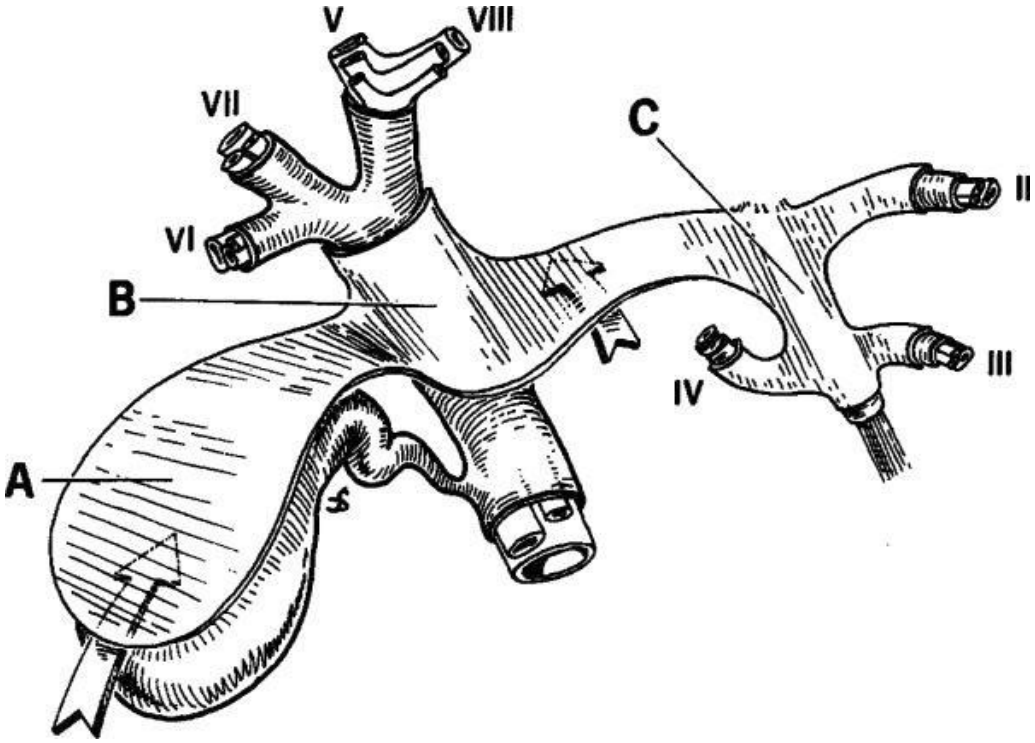


FIGURE 2: the anatomy of the plate system. cystic plate (A) above the gallbladder, the hilar plate (B) above the biliary confluence and at the base of the quadrate lobe, and the umbilical plate (C) above the umbilical portion of the portal vein.

BODY AND FUNDUS:

The body of the gall bladder normally lies in contact with the liver surface.it lies anterior to the 2nd part of the duodenum and the right end of the transverse colon. The fundus lies at the lateral end

of the body and usually projects past the inferior border of the liver to a variable length. It often lies in contact with the anterior abdominal wall behind the 9^{th} costal cartilage where the lateral edge of the right rectus abdominus crosses the costal margin. This is the location where enlargement of the gall bladder is best sought on clinical examination.

The fundus of gall bladder may be folded back upon the body of gall bladder: PHRYGIAN CAP.

EXTRAHEPATIC BILIARY TREE

CYSYIC DUCT

The cystic duct is about 3 to 4 cm in length, passes posteriorly to the left from the neck of gallbladder, and joins the common hepatic duct to form the common bile duct. It almost runs parallel to it and is adherent to common hepatic duct for a short distance before joining it.

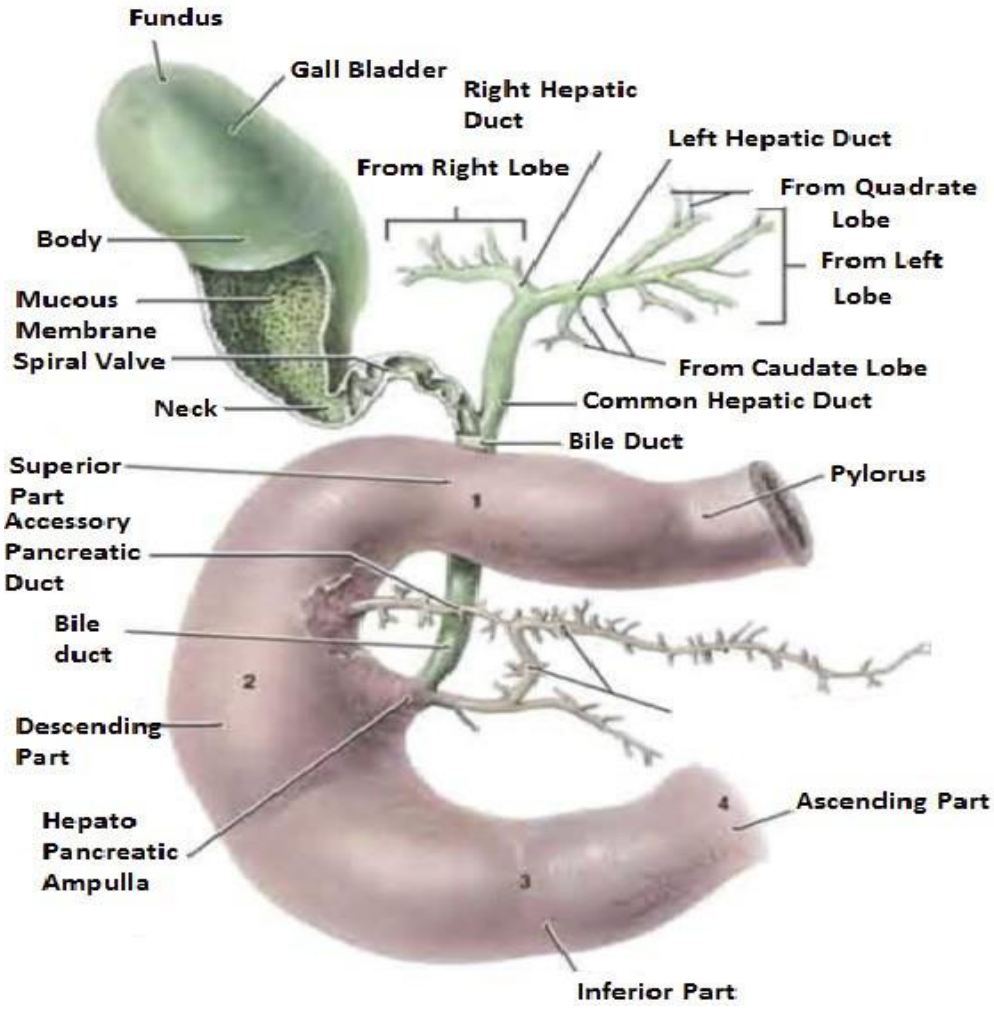


FIGURE 3: showing the anatomy of the gallbladder, biliary radicals, pancreatic duct and the hepatopancreatic ampulla.

ANATOMICAL VARIATIONS OF CYSTIC DUCT

1) The cystic duct occasionally drains into the right hepatic duct in which case it may be elongated, lying anterior or posterior to CHD and joins the right hepatic duct on its left border.

2) The cystic duct lies along the right edge of the lesser Omentum, all the way down to the level of the duodenum before the junction is formed. Here cystic duct and common bile ducts are usually closely adherent.

3) The cystic duct may be double or absent in which case gall bladder drains directly into common bile duct.

4) One or more accessory hepatic ducts occasionally emerge from segment V of the liver and joins either the right hepatic duct the common hepatic duct, the common bile duct, the cystic duct or the gall bladder.

They project obliquely in regular succession, appearing to form a spiral valve when the duct is cut in longitudinal section. When the duct is distended the spaces between the folds dilate and externally it appears twisted like the neck of the gallbladder.

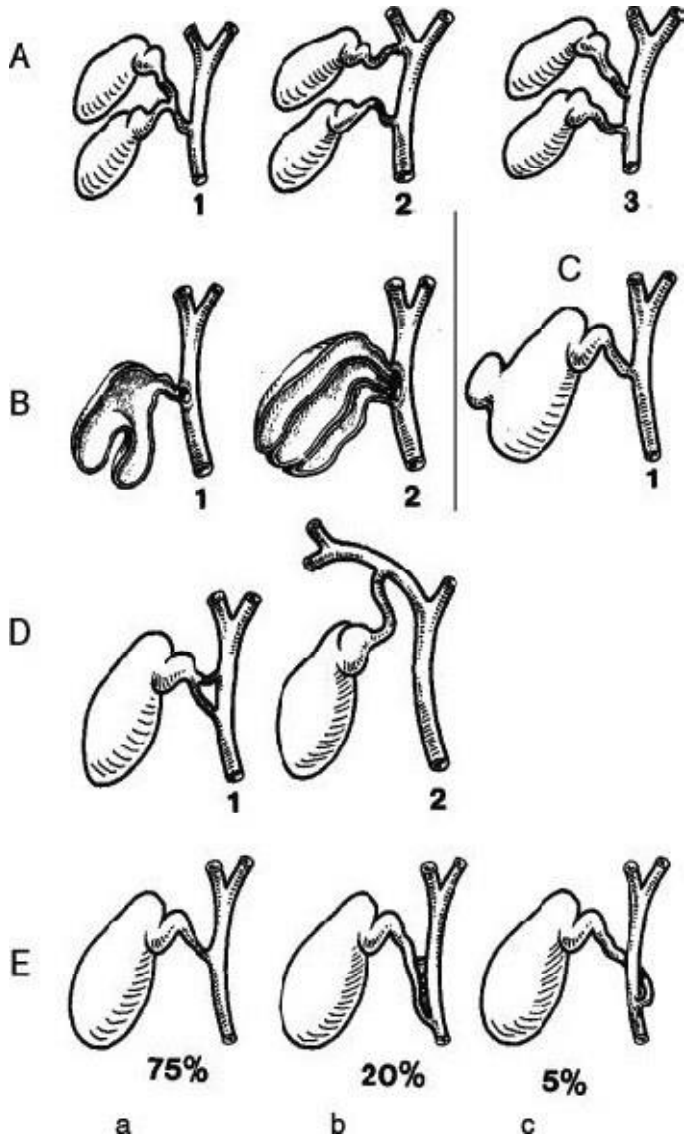


FIGURE 4: variations in gallbladder and cystic duct anatomy: duplicated gallbladder (A), septum of the gallbladder (B), diverticulum of the gallbladder (C), variations in cystic ductal anatomy (D). Different types of union of the cystic duct and common hepatic duct (E).

HEPATIC DUCTS

The main right and left hepatic ducts emerge from the liver and unite near the right end of the porta hepatis as the common hepatic duct. This descends for about 3 cm before joining cystic duct at an acute angle to form common bile duct.

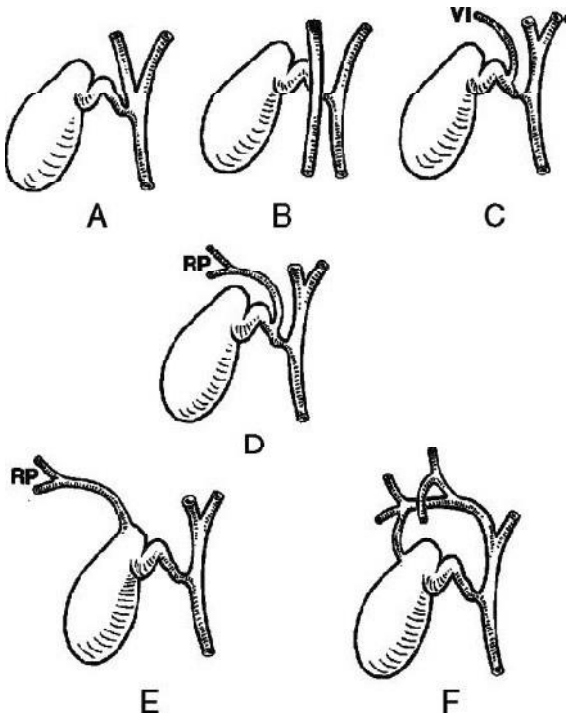


FIGURE 5: The variations of ectopic drainage of the intrahepatic ducts into the gallbladder and cystic duct. A, cystic duct into the biliary confluence. B, cystic duct into the left hepatic duct associated with no biliary confluence. C, segment VI duct into the cystic duct. D,right posterior sectorial duct into the cystic duct. E, distal part of the right posterior sectorial duct into the neck of the gallbladder. F, proximal part of the right posterior sectorial duct into the body of the gallbladder.

COMMON BILE DUCT

Common bile duct is formed near the porta hepatis, by the junction of the cystic and common hepatic ducts.it is usually between 6 and 8 cm in length and about 6 mm in diameter in adults.it descends posteriorly and to the left, anterior to epiploic foramen, in the right border of lesser omentum. It lies anterior and to the right of portal vein and to the right of the hepatic artery. The duct may lie close to the medial wall of the second part of the duodenum or as much as 2 cm from it.

HEPATOPANCREATIC AMPULLA (OF VATER)

It is formed by the union of CBD and pancreatic duct before entering the 2^{nd} part of the duodenum. Circular muscles usually surround the lower part of the CBD(bile duct sphincter),and frequently also surround the terminal part of the main pancreatic duct(pancreatic duct sphincter)and the hepatopancreatic ampulla(sphincter of oddi).

CALOT'S TRIANGLE - CHOLECYSTOHEPATIC TRIANGLE

The near triangular space formed between the cystic duct, common hepatic duct and the inferior surface of the segment V of the liver is commonly referred to as Calot's triangle. It is enclosed by double layer of peritoneum which forms the short mesentery of the cystic duct, it is perhaps better described as a pyramidal space with one apex lying at the junction of the cystic duct and fundus of the gallbladder, one at the porta hepatis and two closer apices at the attachment of GB to the liver bed. The base the triangle thus lies on the inferior surface of the liver.

CONTENTS OF THE CALOT'S TRIANGLE

1) Cystic artery.

2) Cystic lymphnode.

3) Lymphatics from the GB.

4) 1 or 2 cystic veins.

5) Autonomic nerves to the GB.

6) adipose tissue.

7) May contain any accessory ducts which drain into GB from liver.

VASCULAR SUPPLY AND LYMPHATIC DRAINAGE

CYSTIC ARTERY

The cystic artery usually arises from the right hepatic artery. It usually passes posterior to the common hepatic duct and anterior to the cystic duct to reach the superior aspect of the neck of the gallbladder. It divides into superficial and deep branches, superficial branches ramifies on the inferior aspect of the gallbladder, the deep branches on the superior aspect. These arteries anastomose over the surface of the body and fundus. The cystic artery is an end artery and its occlusion is followed by the gangrene of the gall bladder.

ANATOMICAL VARIATIONS

1) May arise from common hepatic artery, sometimes from the left hepatic artery or rarely from the gastro duodenal or superior mesenteric arteries. In this case it may cross anterior (or less commonly posterior) to CBD or CHD to reach gallbladder.
2) An accessory artery may arise from the common hepatic artery or one of its branches.
3) The cystic artery often bifurcates close to its origin to give rise to 2 arteries supplying gallbladder.

4) Multiple fine arterial branches may arise from the parenchyma of the liver(segment IV or V) and contribute to supply the body particularly when the GB is substantially intrahepatic.The cystic artery gives rise to multiple fine branches which supply the common and lobar hepatic ducts and the upper part of the CBD.

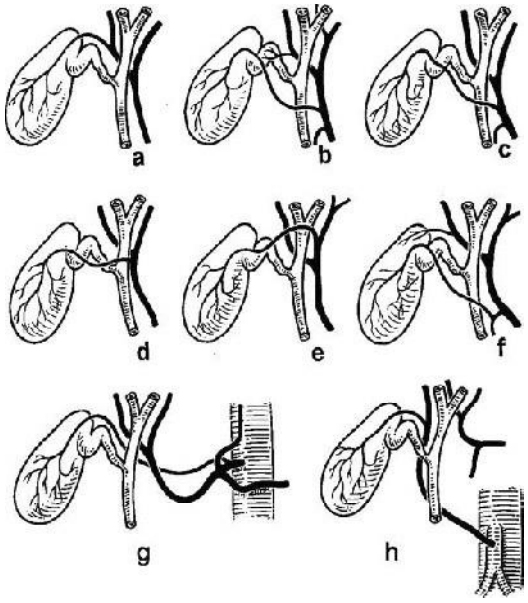


FIGURE 6: variations of the cystic artery: typical course (a); double cystic artery (b); cystic artery crossing anterior to main bile duct (c); originating from the right branch of the hepatic artery and crossing the common hepatic duct anteriorly (d); originating from the left branch of the hepatic artery (e) originating from the gastro duodenal artery (f); arising from the celiac axis (g); originating from a replaced right hepatic artery (h).

DUCTAL ARTERIES:

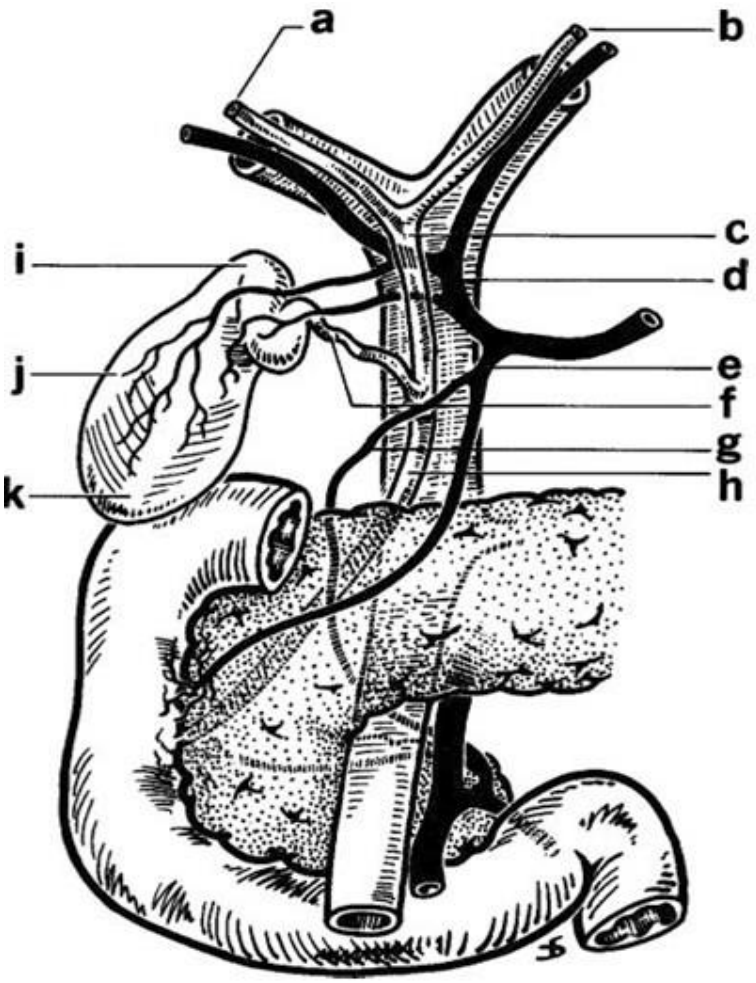


FIGURE 7: Bile duct blood supply. Note the axial arrangement of the vasculature of the supra duodenal portion of the main bile duct and the rich network enclosing the right and left hepatic ducts: right branch of the hepatic artery (a); 9 o'clock artery (b); retro duodenal artery (c); left branch of the hepatic artery (d); hepatic artery (e); 3 o'clock artery (f); common hepatic artery (g); gastroduodenal artery (h).

The common bile duct and hepatic ducts are supplied by a fine network of vessels, which lie in close proximity to the ducts themselves. Disruption of the network during surgical exposure of the bile ducts over a long length frequently causes chronic ischemia and stenosis.Anterior to the CBD, 2 to 4 ascending vessels arise from the retro duodenal branch of the gastro duodenal artery. 3 to 4 descending branches of the right hepatic and cystic arteries arise as this vessels pass close to the lower CHD. These descending and ascending arteries form long narrow anastomotic channels along the length of the duct called medial and lateral trunks.

Posteriorly, a retroportal artery often arises from the coeliac axis, superior mesenteric artery or one of its major branches close to its origin from the aorta. It contributes to the arterial network supplying the supraduodenal part of bile duct system. It runs upward on the posterior surface of the portal vein.

CYSTIC VEINS

Those arising from the superior surface of the body and neck lie in the areolar tissue between the gall bladder and the liver and enter the liver parenchyma to drain into the segmental portal veins. The remainder forms 1 to 2 cystic veins, which enter

LYMPHATICS

Numerous lymphatic vessels run from the submucosal and subserosal plexuses on all aspects of the gall bladder and cystic duct. Those on the hepatic aspect of the gallbladder connect with the intrahepatic lymphatics. The remainder drain into the cystic node, which usually lies above the cystic duct in the tissue of Calot's triangle. This node, and some lymphatic channels which bypass the cystic node, drain into a node lying in the anterior border of the free edge of the lesser Omentum.

INNERVATION

The gall bladder and the extrahepatic biliary tree are innervated by branches from the hepatic plexuses. The retroduodenal part of the CBD also has contribution from the pyloric branches of vagus, which also innervate the smooth muscles of the hepatopancreatic ampulla.

REFERRED PAIN

In common with other structures of foregut origin, pain from stretch of CBD or gallbladder is referred to the central epigastrium. involvement of overlying somatic peritoneum produces pain which is more localized to the right quadrant.

EMBRYOLOGY

The liver primodium appears in the middle of the third week as an outgrowth of the endodermal epithelium at the distal end of the foregut. This outgrowth, the hepatic diverticulum or the hepatic bud consists of rapidly dividing cells that penetrate the septum transversum, that is the mesodermal plate between the pericardial cavity and the stalk of the yolk sac. While the hepatic cells continue to penetrate the septum, the connection between the hepatic diverticulum and the foregut (duodenum) narrows forming the bile ducts.

On day 26, a distinct endodermal thickening appears on the ventral side of the duodenum just caudal to the base of the hepatic diverticulum and buds into ventral mesentery.

This cystic diverticulum will form the GB and the cystic duct. Cells at the junction the hepatic and cystic duct proliferate and form the CBD.In the 10th week of development the weight of liver is approximately 10% of the total body weight due to large number of sinusoids and large nests of proliferating cells, which produce red blood cells and white blood cells. It lies between the hepatic cells and the wall of the vessels.Approximately at 12th week of life liver begins to produce bile, which is dark green in colour.

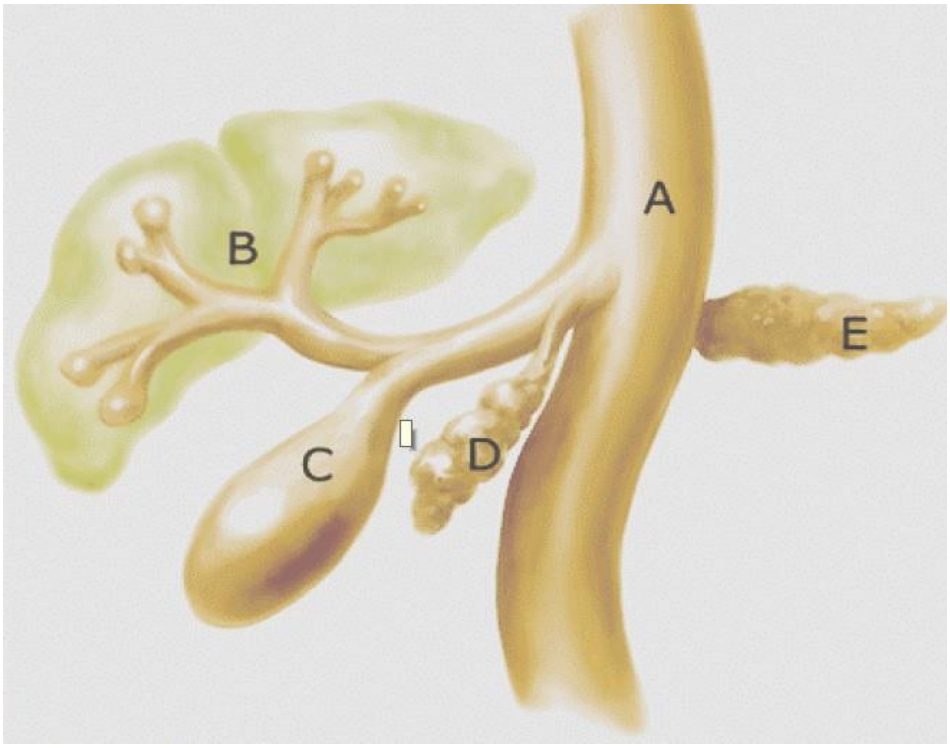


FIGURE 8: illustrating the foregut (A), the cranial end of the hepatic diverticulum which represents Pars hepatica (B) and the Cystic diverticulum (C). The ventral (D) and dorsal (E) pancreas are also demonstrated.

HISTOLOGY

GALLBLADDER

The mucosa is yellowish-brown and elevated into minute rugae with a honeycomb appearance. In section, projections of the mucosa into the gallbladder lumen resemble intestinal villi, but these are not fixed structures and the surface flattens as the gallbladder fills with bile.

The epithelium is a single layered columnar epithelium with apical microvilli. goblet cells are absent. Basally, the spaces between epithelial cells are dilated. Many capillaries lie beneath the basement membrane. Beneath it is a thin fibromuscular layer composed of fibrous tissue mixed with smooth muscles which are arranged loosely in longitudinal, circular and oblique bundles.

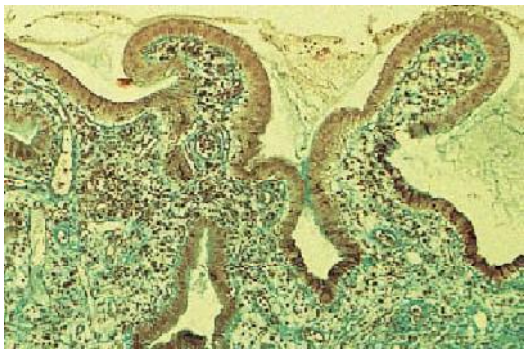


FIGURE 9: Microscopy of gall bladder wall

BILE DUCTS:

The larger biliary ducts have external fibrous and internal mucous layers. The former is fibrous connective tissue which contains variable amount of connective tissue which contain variable amount of longitudinal, oblique and circular smooth muscles. The epithelial covering is columnar and contain many tubuloalveolar mucous glands

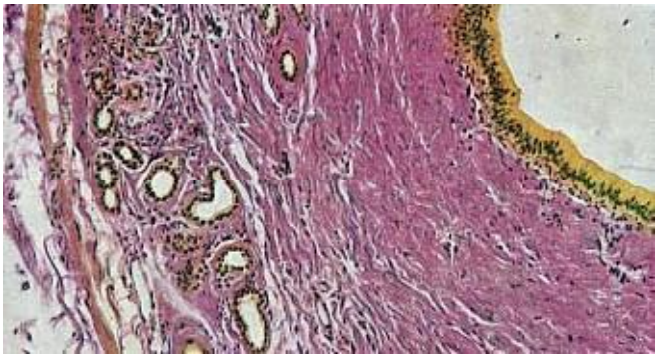


FIGURE 10: Microscopy of common bile duct

PHYSIOLOGY

Bile is made up of bile salts, bile pigments and other substances dissolved in an alkaline medium. About 500 ml is secreted daily. The glucuronides of the bile pigments, bilirubin and biliverdin are responsible for golden yellow colour.

TABLE 1: Composition of hepatic bile

Water	97.0%
Bile salts	0.7%
Bile pigments	0.2%
Cholesterol	0.06%
Inorganic salts	0.7%
Fatty acids	0.15%
Lecithin	0.1%
Fat	0.1%
Alkaline	-------

Entire pool recycles twice per meal and 6 to 8 times per day.

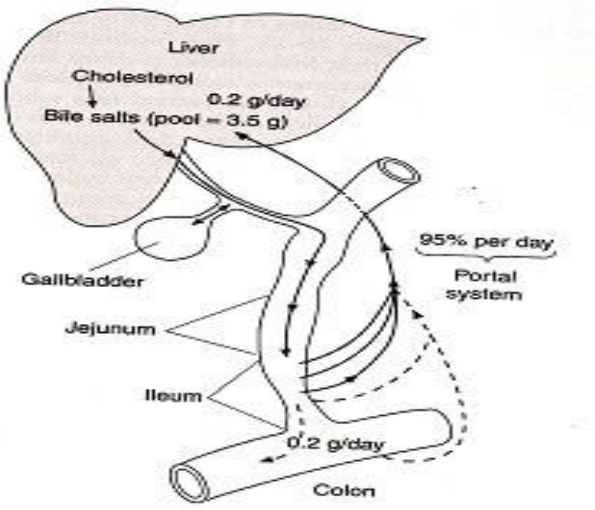


FIGURE 11: Showing enterohepatic circulation of bile salts.

BILIRUBIN METABOLISM AND EXCRETION

Most of the bilirubin in the body is formed by the breakdown of hemoglobin. it is bound to cytoplasmic proteins. It is conjugated to glucuronic acid by UDP-glucuronyl transferase, This diglucuronide is water soluble and is transported actively against concentration gradient into bile canaliculi. A small amount of bilirubin glucuronide escapes into blood, where it is bound to albumin and excreted in urine.The intestinal mucosa is relatively impermeable to conjugated bilirubin but is permeable to unconjugated bilirubin and to urobilinogen. Small amounts of urobilinogen enters the general circulation through portal circulation and is excreted in urine.

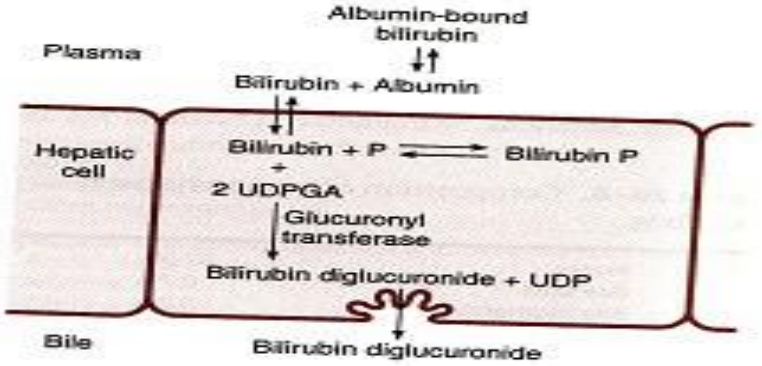


FIGURE 12: Metabolism of bilirubin in liver. p-intracellular binding protein, udpga-uridine diphosphate glucuronic acid, udp-uridine diphosphate.

REGULATION OF BILIARY SECRETION:

The tone of sphincter of Oddi decreases when food enters mouth. Fatty acids and amino acids in the duodenum release CCK, which cause gall bladder contraction. Substances that cause contraction of gallbladder are called cholagogues.

PATHOGENESIS

In the west, about 80% are cholesterol stones, containing more than 50% of crystalline cholesterol monohydrate. The remainder are composed predominantly of bilirubin calcium salts and are designated pigment stones.

CHOLESTROL STONES

Cholesterol is rendered soluble in bile by aggregation with water soluble bile salts and water insoluble lecithin, both of which act as detergents. When cholesterol concentration, exceed the solubilizing capacity of bile (supersaturation)

1) Bile must be supersaturated with cholesterol: this appears to be a primary defect, mediated by abnormal regulation of hepatic mechanisms for delivering cholesterol to bile. The excess free cholesterol is toxic to gallbladder, penetrating the wall and exceeding the ability of the mucosa to detoxify it by esterification. Gallbladder hypo motility ensues. Muscular stasis appears to result both from intrinsic neuromuscular dysmotility and decreased response neuromuscular response to CCK.

2) Gallbladder hypomotility promotes nucleation.

3) Cholestrol nucleation in bile is accelerated: due to shift in balance between antinucleating and pronucleating proteins and presence of micro precipitates of inorganic or organic calcium salts

4) Mucus hypersecretion in the GB traps the crystals, permitting their aggregation into stones.

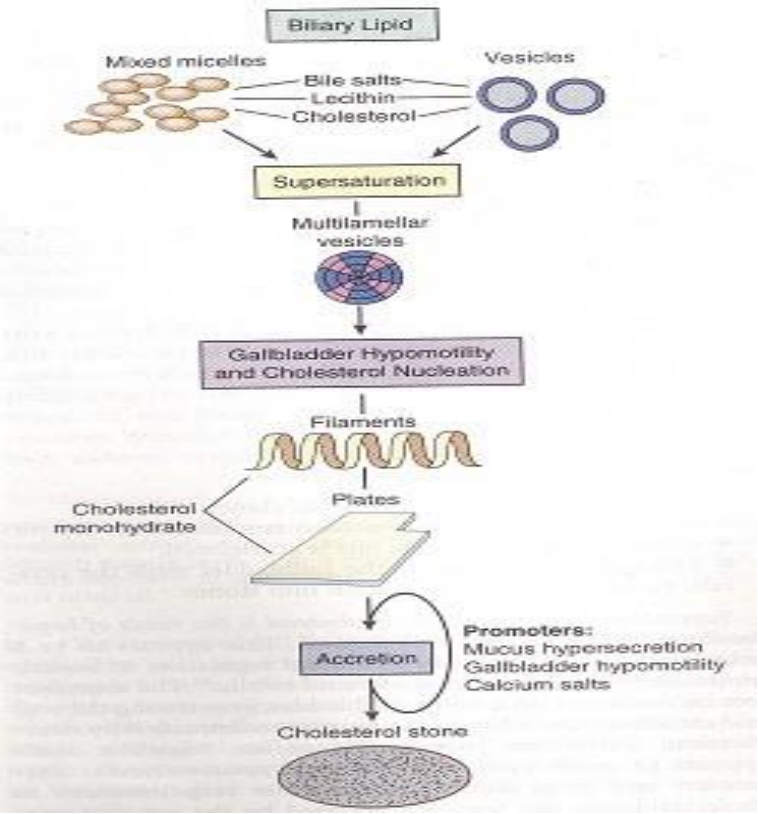


FIGURE 13: Schematic representation of four contributory factors for cholelithiasis: supersaturation, gallbladder hypomotility, crystal nucleation and accretion within the gallbladder mucous layer.

TABLE 2: Superimposed conditions that exacerbate defective GB emptying and cholesterol stone formation

Prolonged fasting	Total parentral nutrition
Pregnancy	Spinal cord injury
Rapid weight loss	

PIGMENT STONES

Pigment stones are complex mixtures of abnormal insoluble calcium salts of unconjugated bilirubin along with inorganic calcium salts. Infection of biliary tract with E.coli or ascaris lumbricoids or by the liver flukes opisthorchis sinensis leads to release of microbial β-glucuronidase, which hydrolyses bilirubin glucuronides to unconjugated bilirubin.

MORPHOLOGY

CHOLESTROL STONES

Arises exclusively in GB and are composed of cholesterol ranging from 100 to 50%. Pure cholesterol stones are pale yellow, round to ovoid and have a fine granular, hard external surface which on transection reveals a glistening radiating crystalline palisade. With increasing proportions of calcium carbonate, phosphates and bilirubin, the stones exhibit discolouration and may be lamellated and gray white to black on transection.

Most often multiple stones are present that range upto several centimeters in diameter. Surfaces of multiple stones may be rounded or faceted, owing to tight apposition. Stones composed largely of cholesterol are radiolucent; sufficient calcium carbonate is found in 10 to 20% of cholesterol stone to render them radiopaque.

PIGMENT STONES

Are classified as black and brown stones. Black pigment stones are found in sterile gallbladder bile, and brown in infected intrahepatic and extrahepatic ducts.

Mucin glycoproteins act as binding proteins in both cholesterol and pigment stones.

THE NATURAL HISTORY OF GALLSTONES

In 1992, it was estimated that 10% to 15% of the adult population in the United States had gallstones, about 1 million patients are newly diagnosed annually. Gallstones are the most common digestive disease

EPIDEMIOLOGY:

Gallstones are most common gastrointestinal illness with a prevalence of 11 to 36% in autopsy reports. Only first degree relatives of the patients with gallstones and obesity (BMI >30 kg/m^2) have been identified as strong risk factors for the development of symptomatic gallstone disease.

TABLE 3: Risk factors for gallstones

Obesity	First degree relatives
Rapid weight loss	Drugs: ceftriaxone,postmenopausal estrogens, total parenteral nutrition
Childbearing	Ethnicity: Native American(Pima Indian) , Scandinavian
Multiparity	Ileal disease, resection or bypass
Female sex	Increasing age

CLINICAL PRESENTATION

Most patients remain asymptomatic from their gallstones. Although the mechanism unclear, some patients develop symptomatic gallstones with biliary colic caused by a stone obstructing the cystic duct. Only 1% to 2% of asymptomatic individuals with gallstones develop serious symptoms or complication related to their gallstones per year; therefore only about 1% require cholecystectomy. Once symptomatic, patients tend to have recurring symptoms, usually repeated episodes of biliary colic. Nonspecific gastrointestinal symptoms develop in 10 to 30% of patients and 5 to 10% of patients develop classic biliary symptoms.

BILIARY COLIC

Acute obstruction of the gallbladder by calculi results in biliary colic, a common misnomer because the pain is not colicky in the epigastrium or right upper quadrant. Biliary colic is a constant pain that builds in intensity and can radiate to the back, interscapular area or right shoulder. The pain is described as a band-like tightness of the upper abdomen that may be

associated with nausea and vomiting. This is due to a normal gallbladder contracting against a luminal obstruction, such as a gallstone impacted in the neck of the neck of the gallbladder, the cystic duct or the CBD. The pain is most commonly triggered by fatty foods, but it can also be initiated by other types of food or even occur spontaneously. An association with meal is present in only 50% of patients, and in these patients, the pain often develops more than 1 hour after eating

INVESTIGATIONS

LIVER FUNCTION TEST

Biliary colic, in the absence of gallbladder pathology or common bile duct obstruction don't produce abnormal laboratory values. Obstructive choledocholithiasis have raised direct bilirubin and elevated alkaline phosphatase levels. Leukocytosis predominantly neutrophils are present in a Cholecystitis and cholangitis.

PT-INR

Prolonged PT is present in liver dysfunction which needs to be normalized before surgery.

ROUTINE BLOOD INVESTIGATIONS

Includes complete haemogram, renal function tests and ECG.

IMAGING STUDIES

PLAIN RADIOGRAPHS

Only about 15% of gallstones contain enough calcium to render them radiopaque and therefore visible on plain abdominal films. Plain films are important are important to exclude perforated ulcer with free intraperitoneal air, bowel obstruction with dilated loops, or right lower lobe pneumonia.

ULTRASONOGRAPHY

An ultrasound is the initial investigation of any patient suspected of disease of the biliary tree. Abdominal ultrasound is a part of routine evaluation in patients withcholelithiasis and has a sensitivity of >98% and sensitivity of >95%.[10] In addition to identifying gallstones, ultrasound can also detail signs of cholecystitis such as thickening of the gallbladder wall, pericholecystic fluid, and

impacted stone in the neck of the gallbladder. Dilation of the extrahepatic (>10 mm) or intrahepatic (>4 mm) bile ducts suggests biliary obstruction.

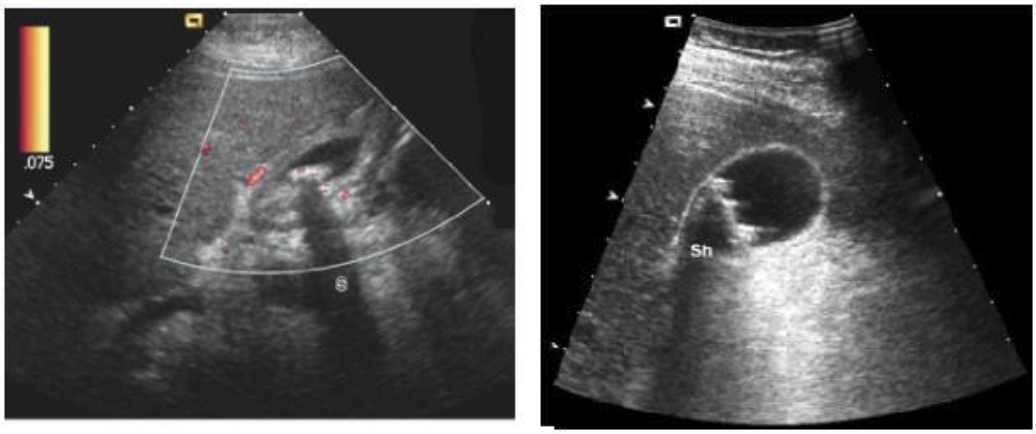


FIGURE 14: A, Echogenic foci in the gallbladder with acoustic shadowing (S) are characteristic of gallstones. In this patient, the gallbladder wall is thickened, but not hypervascular. Features suggest chronic cholecystitis. B, Multiple stones are layered in the dependent portion of the gallbladder, but the wall is not thickened. .

ORAL CHOLECYSTOGRAPHY

Identifies filling defects in a visualized, opacified gallbladder after oral administration of a radioopaque compound that passes into the gallbladder. It is contraindicated in patients with vomiting, biliary obstruction, jaundice, or hepatic failure.

COMPUTED TOMOGRAPHY

CT identifies gallstones within the biliary tree and gallbladder with a sensitivity of only about 55% to 65%. This is because both gallstone and bile are isodense and stones are identified only if they are calcified.

SCINTOGRAPHY

Scintography is useful to visualize the biliary tree, assess liver and gallbladder function. Nonvisualization of the gallbladder at 2 hours after injection is reliable evidence of cystic duct obstruction. Biliary scintography followed by CCK administration is helpful for documenting biliary dyskinesia when gallbladder contraction accompanies biliary track pain in patients without evidence of stones (CCK hepatobiliary 2,6-dimethyl-iminodiacetic acid (HIDA).

INTRAOPERATIVE CHOLANGIOGRAPHY

The first operative cholangiogram was reported in 1936 by Micken. Mirizzi in 1937 performed the first cystic duct cholangiography and this procedure remains the most accepted method for performing (IOC) today.

TECHNIQUES

Cystic duct cholangiography.

Gallbladder cholangiography.

Kumar's technique.

TABLE 4: Indications for routine IOC

Detection of unsuspected CBD stones
To detect anomalous anatomy
Presence of accessory duct
Short cystic duct
Identification of iatrogenic injury

COMPLICATIONS OF GALLSTONES

Acute cholecystitis

Chronic calculus cholecystitis

Choledocholithiasis with or without cholangitis

Gallstone pancreatitis

Gallstone ileus

Gallbladder carcinoma

MANAGEMENT OF CHOLELITHIASIS

The non operative management of gall stones has long fascinated physicians. the idea of dissolving gall stones attracted early interest with Durande in 1782. In 1975, Makino reported gall stone dissolution by administering ursodeoxycholic acid.

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY(ESWL)

ESWL is in use since 1986. It is used to fragment stones. Patient selection is very crucial for success and are selected according to criteria laid down in Munich study.

The criteria are functioning of gall bladder and stone should be

i. Cholestrol stone
ii. Less than 3 in number
iii. Less than 3 cm.

Recurrence rate is 5-7% at 12 months and 15% at 24 months.

MEDICAL MANAGEMENT

Ursodiol (urosodeoxy cholic acid) contitutes less than 5% of total bile salt pool.

CLINICAL USES

1) Dissolution of small cholesterol gallstones in patients with symptomatic gallstones who refuse cholecystectomy or who are poor surgical candidates. At a dosage of 10 mg/kg/day for 12 -24 m, dissolution occurs in upto 50% of patients with small (<5-10 mm)non calcified gallstones.

2) Prevention of gallstones in obese patients undergoing rapid weight loss therapy.

3) At a dosage of 13-15mg/kg/d is helpful for patients with early stage primary biliary cirrhosis, reducing liver function abnormalities and improving liver histology.

ADVERSE EFFECTS

Ursodiol is practically free of serious adverse effects. Bile salt induced diarrhea is uncommon.

PREOPERATIVE PREPARATION

1) Blood coagulation should be normalized in patients with prior, by giving vitamin K (IM in 3 doses)

2) A prophylactic antibiotic either with premedication or at the time of anesthesia induction is given. A second generation cephalosporin is appropriate.

3) Subcutaneous heparin or antiembolic stocking are used to prevent deep vein thrombosis.

OPEN CHOLECYSTECTOMY

Indications for OC

Poor pulmonary or cardiac reserve

Suspected or known gallbladder cancer

Cirrhosis and portal hypertension

Third-trimester pregnancy

Combined procedure

Conversion from laparoscopic approach

A short right upper transverse incision is made centered over the lateral border of the rectus muscle-kocher's incision. The gallbladder is appropriately exposed and packs placed on the hepatic flexure of the colon, the duodenum, and the lesser

Omentum to clear view of the anatomy of the porta hepatis. This packs are retracted using the left hand of the assistant, or a stabilized ring retractor is used to keep the pack in position. A duval forceps is placed on the infundibulum of the gallbladder and the peritoneum overlying calot's triangle is stretched. The calot's triangle is dissected to expose the cystic duct and the cystic artery. These are confirmed by tracing them to enter the gallbladder. The cystic artery is ligated and cut. The cystic duct is then ligated and divided. A suction drain is placed before closure.

When there is doubt about anatomy, a fundus first or retrograde cholecystectomy dissecting on the gallbladder wall down to the cystic duct can be helpful.

LAPAROSCOPIC CHOLECYSTECTOMY

LC is one of the most common surgeries performed and has replaced open cholecystectomy. In 1992, The National Institute of Health (NIH) consensus development conference stated that laparoscopic cholecystectomy "provides a safe and effective treatment for most patients with symptomatic gallstones.

INDICATIONS OF LAPAROSCOPIC CHOLECYSTECTOMY

a)Symptomatic cholelithiasis

i) Biliary colic: Once the patient experience symptoms, there is a greater than 80% chance that they will continue to have symptoms. There is also a finite risk of disease related complications such as acute cholecystitis, gallstone pancreatitis and choledocholithiasis.

ii) Acute cholecystitis.

iii) Gallstone pancreatitis.

b)Asymptomatic cholelithiasis

Patient with asymptomatic gallstone have less than 20% chance of ever developing symptoms, and the risks associated with prophylactic operation outweigh the potential benefit of surgery in most patients. Therfore prophylactic cholecystectomy is recommended in

i) Sickle cell disease

ii) Total parenteral nutrition

iii)Chronic immunosuppression

iv) No immediate access to health care facilities (eg: missionaries, military personal, peace corps workers, relief workers)

v) Incidental cholecystectomy for patients undergoing procedures for other indications.

c) Acalculous cholecystitis or biliary dyskinesia

d) Gallbladder polyps >1 cm in diameter.

e) Porcelain gallbladder.

CONTRAINDICATION TO LAP CHOLECYSTECTOMY

ABSOLUTE

1) Unable to tolerate general anesthesia.

2) Refractory coagulopathy

3) Suspicion of carcinoma

In porcelain gallbladder and potentially curable GB malignancy, due to persistent concerns with adequacy of resection and reports of port site metastasis associated with the use of minimally invasive surgical technique for treatment of intra-abdominal malignancies.

RELATIVE

1) Previous upper abdominal surgery

2) Cholangitis

3) Diffuse peritonitis with hemodynamic compromise

4) Cirrhosis and /or portal hypertension

Brittle, friable liver that may be difficult to retract in cephalad direction, associated coagulopathy and due to abnormal portosystemic venous shunts in portal hypertension.

5) Cholecystoenteric fistula

6) Morbid obesity was a contraindication previously due short trocar length and sheath designs making institution of pneumoperitoneum problematic.

7) Chronic obstructive pulmonary disease

8) Pregnancy

Due to unknown effect of co2 on foetus-therefore avoided in first trimester. Open insertion of port or location of initial port in right upper quadrant to avoid damage to uterus. Maintenance of pneumoperitoneum to <12 mm of hg and maternal hyperventilation with monitoring of pco2 is needed to avoid fetal acidosis.

PATIENTS LIKELY TO REQUIRE CONVERSION

a) Multiple prior operations-due to difficulty in safe access to peritoneal cavity.

b) Acute severe cholecystitis: Due to difficult dissection secondary to inflammation, adhesions or edema.

c) Acute pancreatitis: Difficult visualization of calot's triangle due to edematous pancreatic head.

d) Abnormal anatomy: Higher likelihood of biliary/vascular injury.

e) Cirrhotic liver: Higher likelihood of liver injury and haemorrhage.

f) Third trimester pregnancy: Higher likelihood of uterine injury during access.

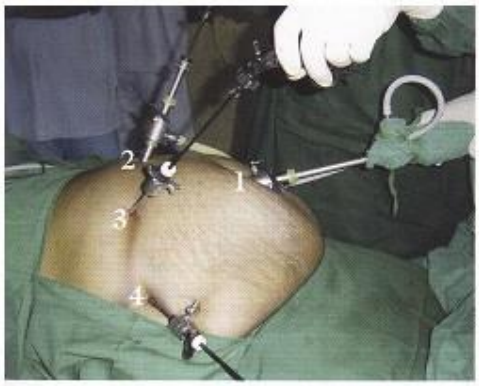
g) Morbid obesity: Difficulty in access and dissection.

h) Evidence of generalized peritonitis.

i) Septic shock from cholangitis.

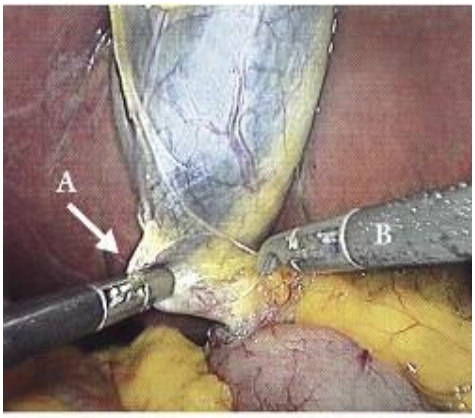
FIGURE 15: Showing steps of laparoscopic cholecystectomy

STEP 1: Patient Position

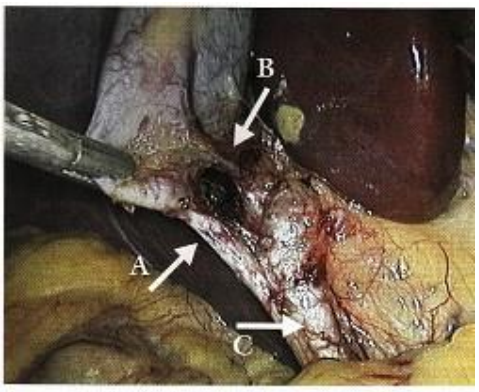


STEP 2: Port Placement

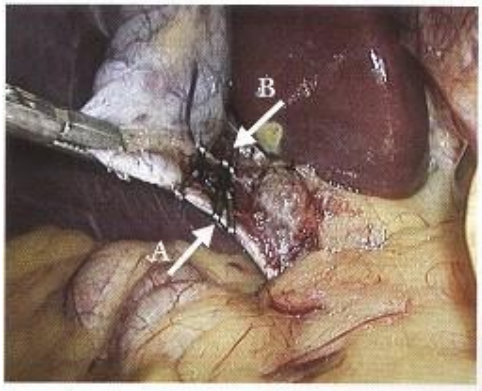
STEP 3: Exposure of Porta Hepatis



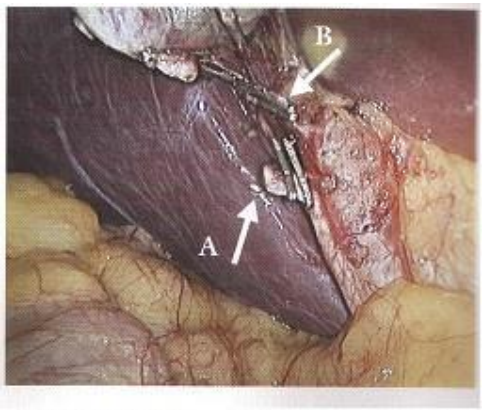
STEP 4 : Dissection of Calot's Triangle



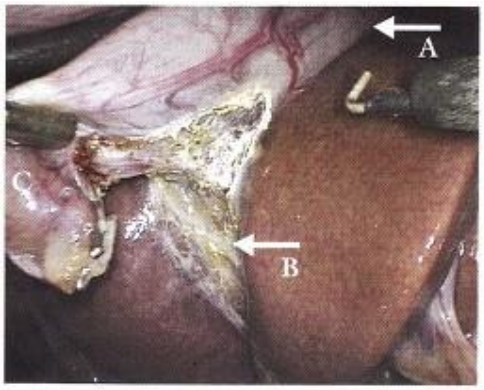
STEP 5: Identification of Cystic Duct(A), Cystic Artery(B) and CBD(C)



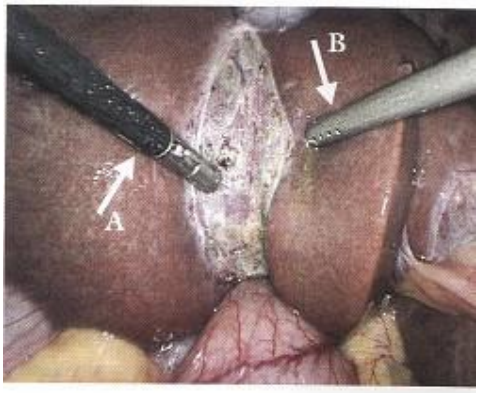
STEP 6: Clipping of Cystic artery and Cystic duct



STEP 7: Division of Cystic artery and Cystic duct



STEP 8: Detachment of GB from the Liver bed



STEP 9: Inspection of Liver bed

STEP 10: Etraction of GB

STEP 11: Extraction of the entire unit

APPROACHES

A) NORTH AMERICAN APPROACH

The patient is kept in supine in antitrendlenburg position(15 degree head up tilt) with left lateral tilt (15-20 degree).this ensures that the bowel and Omentum falls down and medially, away from the operative site. The operating surgeon and camera surgeon stand on the left of the patient while the assistant surgeon stands on the right of the patient. Two monitors are placed at 10’0 and 2’0 clock position.

PORT PLACEMENT

Ports are placed by screwing motion. Second hand is used to prevent inadvertent plunge of the trocar. The assistant should provide counter traction on the abdominal wall during placement of the first trocar.

10 mm port is placed in the midline, usually through the umbilicus. Sub-umbilical position preferred in patients with cirrhosis due to the presence of dilated, tortuous anastomotic veins in the periumbilical region, visceroptic liver, hepatomegaly and in patients with pendulous abdomen.

If a previous abdominal surgery has been performed through a vertical midline incision, abdomen in insufflated through a site adjacent to the umbilicus, and a primary 5 mm trocar is placed in the right upper quadrant. The 10 mm trocar is then placed under direct vision, avoiding the adhesions from previous operation, under direct vision through a 5 mm telescope passed through 5 mm port.

Pneumoperitoneum is created through Hasson technique if previous surgery prevent primary puncture through the umbilicus

Another 10 mm port is placed in the epigastrium starting from the midline and angling toward the gallbladder, at the level of the inferior edge of the liver and to the right of falciform ligament. If it is placed too high, segment IV of the liver will impede the ability to get to the gallbladder.

A 5 mm trocar is placed 2 to 3 cm below the costal margin in the midclavicular line. The fourth, a 5 mm trocar is generally placed in the anterior axillary line, several centimeters below the fundus of the gallbladder, but its position is variable.

B) FRENCH/EUROPEAN APPROACH

The patient is in semi lithotomy antitrendlenburg position with leg in allen stirrups such that the thighs are almost parallel to the ground. The operating surgeon stands between the legs of the patient with the camera surgeon on the right of the patient and the assistant on the left of the patient.

PORT PLACEMENT

A camera port is placed at umbilicus, 5 mm epigastric port is placed to allow retraction by assistant, 10 mm right hand working port is placed in left hypochondrium or in the midline between the camera port and the epigastric port and the left hand working port (5 mm) is placed in the right hypochondrium.

ADDITIONAL PORT

a) Left lumbar 5 or 10 mm port for three prong or flat blade retractor for downward traction of the colon, Omentum and duodenum. This maneuver gives wide exposure of the hilum.

b) 5 mm port midway between epigastric and right midclavicular ports for lifting the quadrate lobe using blunt tipped retractors (French technique), eg in liver cirrhosis, left lobe gallbladder.

PNEUMIOPERITONEUM

LC is generally performed with a carbondioxide pneumoperitoneum at a pressure of 15 mm of Hg pressure. Other gases like nitrogen oxide, helium and argon are being tried.

TECHNIQUES:

a) VERESS NEEDLE TECHNIQUE

In veress needle technique; pneumoperitoneum is generally created by sliding a veress needle through the umbilicus,confirming its position by allowing saline to run through the needle from a plungerless syringe, and then attaching the needle to tubing from carbon dioxide. Once it is confirmed to be intra-abdominal, the flow rate can be increased until 15 mm Hg of pressure is attained.

b) OPEN (HASSON) LAPAROSCOPY TECHNIQUE

In open technique, abdominal cavity is entered under direct vision. Once the peritoneal cavity is entered, the initial trocar is inserted and its position is secure with two stay sutures. The abdominal cavity can then be insufflated with carbon dioxide.

STEPS

A) PATEINT PREPARATION,EQUIPMENT AND ANAETHESIA

EQUIPMENT

a) High-quality videoscope with a 300 w light source be coupled to two high-resolution monitors.

b) High-flow carbon dioxide insufflator.

c) Four trocars: 2-10 mm trocars and 2-5 mm trocars.

d) Hand instruments: Monopolar electrode c-hook with suction and irrigation, a fine tipped dissector, two gallbladder grasper, a large gallbladder extractor, scissors and a hemoclip applier.

e) 10 mm stone retrieval grasper.

f) Micro scissor, a specialized cholangiogram clamp and a 4 or 5 mm French catheter to perform cholangiogram.

ANAESTHESIA TECHNIQUE

Generally, nitric oxide is avoided to minimize the likelihood of bowel distention. Intravenous fluids must be run frugally as the insensible fluid losses through the closed abdomen are minimized and pneumoperitoneum is a strong stimulator of antidiuretic hormone. End tidal pco2 is monitored to check for hypercarbia and acidosis secondary to carbon dioxide pneumoperitoneum.

Narcotics are used in smaller doses and powerful antiemetic is used to lessen postoperative nausea. Once the patient is anesthetized and intubated, a foley catheter, sequential compression devices and orogastric tube are generally placed.

North American approach is generally followed.

B) EXPOSURE OF PORTA HEPATIS

The fundus of the gallbladder is held with a ratchet grasper and retracted by the assistant in a cranial direction, which lifts the right lobe of the liver and exposes the calots triangle and hilum of the liver. Adhesions to the underside of the liver and bladder are

carefully taken down beginning near the fundus and proceeding down towards the neck.

C) DISSECTION OF THE CHOLECYSTOHEPATIC TRIANGLE(CALOTS TRIANGLE)

In tensely distended GB, it may be decompressed in two ways- percutaneous verees needle aspiration or the midclavicular trocar is introduced into the fundus of the gallbladder directly and content aspirated.An atraumatic non looking grasper is introduced through the left hand working port to hold the infundibulum and retract it downwards and to the right.using a Maryland's forceps introduced through the epigastric port, the peritoneum of the infundibulum is held and breached by using small bursts of cautery current. Peritoneum on anterior and posterior aspect are stripped down. The infundibular grasper in moved inferolaterally and superomedially (flag technique) to aid the dissection of anterior and posterior surface of calot's triangle.

D) IDENTIFICATION OF THE CYSTIC DUCT AND ARTERY

Methods for ductal identification in laparoscopic cholecystectomy

i) Infundibular or infundibular-cystic technique: In this method the cystic duct is isolated by dissection on the front and the back of the triangle of callot's and once isolated it is traced on to the gallbladder.

ii) Critical view of safety triangle: method requires complete dissection of the cholecystohepatic triangle and separation of the base of the gallbladder infundibulum from the liver bed. When this view is achieved, the two structures entering the gallbladder can only be cystic duct and artery. It is not necessary to see the common bile duct.

Cystic duct is identified at the junction of gallbladder (SAFETY ZONE) and followed down for adequate length for cholangiography. It is not necessary to identify and dissect cystic duct CBD junction (DANGER ZONE).

Cystic artery is identified along with its anterior and posterior branches by blunt dissection. The cystic node sometimes overlies the cystic artery.Both the cystic duct and artery are clipped, two clips on the cystic duct side and one on the gallbladder side. Before clipping the cystic duct the stones in the cystic duct are milked back to GB,Artery is divided before the duct but in certain cases duct is divided first to give exposure to the artery.In case of an impacted cystic duct stone, the cystic duct is clipped at its junction with GB and a partial cut is made just distal to the clip and impacted stone milked back and extracted.

E) DETACHMENT OF GB FROM THE LIVER BED

The GB can be detached from the liver bed using a spatula with monopolar cautery, hook with monopolar cautery, scissors with monopolar cautery or harmonic scalpel. Care should be taken to stay away from the porta hepatis and the liver bed and to avoid perforation of the gallbladder. Traction and counter traction facilitate dissection.Any inadvertent spillage of bile or stones from the GB during the procedure should be immediately controlled by applying clips, pre-tied loops or reapplying the grasping clamp.

Spilled bile is immediately sucked and stone removed.Prior to complete detachment of the gallbladder, the liver bed is inspected for adequate hemostasis or bile leak.The cystic duct remnant and cystic artery stumps are examined. Minor oozing from liver bed is controlled with cauterizing and continuous suction irrigation. Once complete hemostasis is achieved GB is separated completely.

F) EXTRACTION OF THE GB

Extraction of the GB can be done through umbilical or epigastric port. Epigastric port is preferred because:

i) No need to change camera port.

ii) Facilitates thorough rinsing to avoid port tract infection

iii) By extending skin incision, the fascial opening can be easily dilated and majority of GB extracted

iv) Fascial opening closed easily by cutaneous approach.

v) Better cosmetic appearance.

A claw shaped gallbladder extraction forceps is introduced and used to grasp the neck of the GB. If GB is too distended

the neck is pulled out through the skin incision, small nick made and bile suctioned and stones crushed using sponge holder.If the GB is thick preventing its extraction the fascial incision is enlarged using a closed Robert's clamp or extending it.Infected or necrotic GB or a GB with suspicion of carcinoma is placed in a sterile bag before extraction to reduce port site infection.

G) FINAL INSPECTION AND IRRIGATION

After GB is extracted, the epigastric port is replaced and surgical site inspected for bleeding. A thorough wash is given to the GB bed, Morrison's pouch, paracolic gutter and perihepatic areas with saline which is later suctioned.

Venous ooze is controlled from the liver bed by

i)gelatin sponge soaked in hemostatic solution. eg: hemlock solution.

ii) Use of harmonic ball application.

iii) Rarely intracorporeal suturing.

iv) Argon plasma coagulator

H) DRAINGE AND CLOSURE

CLOSURE

If drain is needed a 14 F Redivac tube is placed through 5mm trocar site-lateral most port. Trocars are removed under direct vision to check for bleeding from trocar site. Pneumopritoneumevacuated and subcuticular stitch/skin clip/dermabond.

COMPLICATIONS

A) HEMORRHAGE

i)TROCAR SITE BLEEDING

Trocar site bleeding can be prevented by control of bleeding following skin incision and before inserting trocar.

Any subcutaneous vessel in subcutaneous tissue should be avoided during insertion.

Detection:

the blood may run down the abdominal wall or drip down the instruments into the operative field.

Management: pressure over the site of bleeding by tilting the trocar.

Injection of epinephrine 1:10000 in the vacinity of the bleeding site.

Screwing in the anchoring device of a disposable trocar may compress and stop the bleeding. Suture ligation.

ii) HEMORRHAGE DUE TO BLUNT DISSECTION OF ADHESIONS can be managed with electrocautery

iii) SUDDEN AND PULSATILE BLEEDING IN CALOT'S TRIANGLE

Bleeding in the calot's triangle can be prevented by careful dissection and proper application of clip to cystic artery.

Management: Retraction of the GB is released and the GB is gently pushed into the calot's triangle to obtain temporary respite during which additional port is placed between the umbilical and the epigastric ports.by repeated suction and irrigation, the blood is cleared from the operative field and the bleeding vessel is precisely identified and clipped.

iv) GALLBLADDER FOSSA BLEEDING

GB fossa bleeding can be controlled by electrocautery, packing the site with hemlock soacked gel foam, figure of eight stitch in case of spurter from liver parenchyma.

b) PERFORATION OF GB

GB perforation seen in acute cholecystitis and while detaching GB from the liver bed. This can be prevented by confining to the areolar tissue between the GB and the liver bed during dissection and decompression of the gall bladder if distended.

TABLE 6: Clinical presentation secondary to gallstone spillage

INFECTIVE	CUTANEOUS	MECHANICAL
Liver abscess	Sinus	Intestinal obstruction
Retrohepatic abscess	Port tract infection	
Subhepatic abscess	Granuloma formation	
Retroperitoneal abscess	Colocutaneous fistula	
Loin abscess		
Pelvic abscess		

Management:

Copious irrigation and suction will remove majority of small stones while larger ones are removed using laparoscopic tissue pouch. Drainage catheter is placed. Perforated site must be closed with pretied ligature or by holding with the grasper.

c) DIFFICULTY IN EXTRACTION OF THE GALLBLADDER

seen in gallbladder containing large stones and those with thick wall. In GB containing large stones, the GB is placed in an endobag,the neck retrieved out through the abdomen and stones are crushed and removed. In GB with thickened wall ,the GB is placed in an endobag and extracted.

d) OCCULT CARCINOMA

In cases suspected to have carcinoma intraoperatively, frozen section is sent and if frozen section is positive for carcinoma, then conversion to open technique is considered and radical surgery with excision of port sites done.

e) POST OPERATIVE BILE LEAK

Post operative bile leak can occur due to injury to the CBD, the right hepatic duct or accessory bile duct.This can be prevented by correct identification of the cystic duct and artery, minimum use of electrocautery in calot's triangle dissection and appropriate choice of laparoscopic subtotal cholecystectomy.Postoperative bile leak should be suspected in patients with fever, tachycardia and upper abdominal pain and tenderness persisting or appearing unexpectedly. The diagnosis can be confirmed by USG or ERCP.

If drain is placed most of the minor leak will heal with expectant mangement. In some persistent cases, it may be advisable to decrease the intraductal pressure by nasobiliary drainage, endoscopic spincterotomy or transpapillary stenting.

f) BILE DUCT INJURY

Incidence of CBD injury during LC exceeds that of open cholecystectomy ie 0.5% vs 0.2%.[21] Reasons for the increase in injury during LC included loss of haptic information, incorrect traction forces to the gallbladder, and injudicious use of cautery inside of the triangle of calot.

Risk factors that increase the risk of CBD injury include acute cholecystitis, aberrant anatomy. The most common anatomic variant is an aberrant right hepatic duct.

PREVENTION

i) use a 30 degree laparoscope and high-quality imaging equipment.

ii)Apply firm cephalic traction to the fundus and lateral tractionto the infundibulum so that the cystic duct is perpendicular to the CBD.

iii) Dissect the cystic duct where it joins the gallbladder.

iv) Expose the “critical view of safety” prior to dividing the cystic duct.

v) Convert to open procedure if the infundibulum cannot be mobilized or bleeding or inflammation obscures the triangle of calot.

vi) Perform routine intraoperative cholangiography. It is managed by biliary enteric anastomosis. This is to prevent cholangitis and biliary strictures.

g) BOWEL INJURY

Injury to bowel can occur during trocar insertion or dissection in the right upper quadrant, especially when using electrosurgical devices. The jejunum, ileum and colon can be injured by veress needle and trocars while duodenum is likely to be injured during dissection. Any structure fixed to the undersurface of the umbilicus like the urachus or a meckel's diverticulum is more susceptible to injury during access. The rate of bowel injury between 0 and 0.4% has been reported in various studies.

h) WOUND INFECTION AND INCISIONAL HERNIA

The risk of wound infection following laparoscopic cholecystectomy is less than 1% and the risk of incisional hernia is 0.5%.[23] Use of a retrieval bag for extraction of GB and closure of all port sites larger than 8mm may avoid these complications.

i) DIAPHRAGMATIC INJURY

j) PANCREATITIS

k) PNEUMOPERITONEUM RELATED COMPLICATIONS

Pneumoperitoneum related complications include carbon dioxide embolism, vasovagal reflex, cardiac arrhythmias and hypercapnia acidosis.Hypercapnia and acidosis are due to absorption of carbon dioxide from the peritoneal cavity. Sudden increases in $Paco_2$ may be related to port slippage and extraperitoneal or subcutaneous diffusion of co_2. It is managed by desufflating the abdomen for 10 to 15 min. If reinsufflation results in recurrent hypercapnia, then change the insufflations gas or convert to open.Carbon dioxide embolism is characterized by unexplained hypotension and hypoxia. Characteristic mill wheel murmur is detected on auscultation. This is produced due to the contraction of right ventricle against the blood gas interface.There is an exponential decrease in end tidal co_2 due to complete right ventricular outflow obstruction. It is managed by immediate evacuation of pneumoperitoneum and placement of the patient in left lateral decubitus, head down (Durant) position. This allows the co_2 bubble to float to the apex of the right ventricle, where it is less likely to cause right ventricular outflow obstruction. Patient is hyperventilated with 100% oxygen.

CONVERSION

In 5-10% of cases, conversion to open cholecystectomy may be needed for safe removal of gallbladder, the risk factors for conversion were male sex, obesity, cholecystitis and choledocholithiasis.

ADVANTAGES AND DISADVANTAGES OF LC

COMPARED TO OC

TABLE 9: Advantages and disadvantages of LC compared to OC

ADVANTAGES	DISADVANTAGES
Less post operative pain	Lack of depth perception
Smaller incision	View controlled by camera operator
Better cosmesis	More difficult to control hemorrhage
Shorter hospitalization	Decreased tactile discrimination (haptics)
Earlier return to full activity	Potential co2 insufflation complications
Decreased total costs	Adhesions/inflammation limit use
	Slight increase in bile duct injury

RISK FACTORS OF DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY

i) CLINICAL RISK FACTORS

Stocky male patients due to difficulty in initial port placement

Multiparous women with flabby abdomen due to thinned out

Previous upper abdominal surgery

Cirrhosis of liver

Present or previous acute cholecystitis or acute severe pancreatitis

Previous treatment: percutaneous drainage / cholecystostomy

II) ULTRASOUND CRITERIAS

Thick walled gallbladder(>4 mm)

Contracted (nonfunctioning) gallbladder

Packed stones and large calcified GB.

Polyp or mass lesion without acoustic shadow

Evidence of acute cholecystitis:-impacted stones

Edematous gallbladder wall

Pericholecystic fluid collection

Emphysematous cholecystitis

Subphrenic collection

Intraperitoneal fluid collection due to perforated GB

Fatty liver with hepatomegaly

Cirrhosis of liver

Portal vein thrombosis with cavernoma

SAFETY MEASURES

Selective open technique of pneumoperitoneum

Intraoperative cholangiography to identify biliary anatomy and the CBD stones.

Laparoscopic ultrasound is useful in mapping biliary and vascular anatomy and is superior to operative cholangiogram.

Adequate instrumentation:

i) Toothed graspers to grasp and retract thick walled gallbladder.

ii) Specialized needle drivers and holders

iii) Five pronged retractors.

Hydrodissection

Preliminary decompression

Additional ports for retraction to get adequate exposure

Caudal traction of the hepatoduodenal ligament using multipronged retractor. The port is placed in the left midclavicular line, midway between the camera port and the epigastric port.

Dipping retractor for quadrate lobe lifting (French technique)

PROBLEMS IN DIFFICULT CHOLECYSTECTOMY

ACCESS PROBLEMS

a) ADHESIONS

Post-operative adhesions: In lower abdominal scars, the veress needle is inserted at the site of proposed epigastric port. The umbilical port is inserted under visual guidance. In open appendicectomy scar, Hasson method is the ideal technique for creating pneumoperitoneum. In case of upper abdominal scars present in the midline or right Para median position, the left subcostal veress needle insertion(palmer's point) is used to create pneumoperitoneum. Conversion rate as high as 25% has been reported in patients with extensive upper abdominal adhesions.

Inflammatory adhesions: is usually due to acute cholecystitis or acute severe pancreatitis. These adhesions can easily be removed using suction nozzle. But if the adhesions are organized then sharp dissection is done.

b) INCISIONAL HERNIA

In cases of lower abdominal incisional hernias, appropriate repair could be accomplished after completing laparoscopic cholecystectomy either by open or laparoscopic technique.

c) OBESITY

The veress needle insertion and the insertion of first trocar is difficult. Cystic artery and cystic duct are covered with thick fat hence dissection is difficult.

d) CIRRHOSIS

Due to adhesions with increased vascularity, difficult traction of liver, inadequate exposure of hilum, high risk of GB bleed and high risk hilum.

CONCOMITANT PATHOLOGY

a) MUCOCOELE

Mucocoele is difficult to retract and apply grasping forceps. It is managed by decompression of the GB, using toothed forceps for retraction of GB, removal of the impacted stone either by dislodging into the GB or through an incision over the cystic duct after applying distal clip.

b) GANGRENOUS GB

Due to difficulty in grasping, loss of tissue plane, difficulty in exposure of calot's triangle, performance of intraoperative cholangiogram is difficult, spillage of stones and infected bile; gangrenous GB is difficult to operate.

c) EMPYEMA

d) SCLEROATROPIC GB

The GB is contracted, fibrosed and densely covered with extensive adhesions. Adhesions of the duodenum and the colon are very common and access to calot's triangle is difficult due to fibrous scarring.

e) MIRRIZZI'S SYNDROME

LC is difficult in Mirrizzi's syndrome due to contracted GB with extensive adhesions, CBD may be mistaken for cystic duct and chances of CBD injuries are more and if fistula is not recognized during surgery, biliary peritonitis may occur.

f) PORCELAIN GB

The prevalence of porcelain GB in cholecystectomy specimen ranges from 0.06% to 0.8%.[30] Decompression of the gallbladder and traction is difficult due to calcified wall. Toothed forceps can be used for cranial traction of the GB. Calcification of the cystic duct may require endosuturing or application of endoloops to the cystic duct.

g) CHOLECYSTOENTERIC FISTULAS

Cholecystoenteric fistula is an incidental finding in 0.5 to 0.7% of cases of laparoscopic cholecystectomy for biliary disease.The diagnosis suspected by the presence of air in GB. Problems arise due to difficulty in identification of the anatomy, difficulty in performing cholangiography and due to the requirement of intracorporeal suturing for closure of perforation.

h) ACUTE BILIARY PANCREATITIS

Difficulty in performing LC in acute biliary pancreatitis is due to-extensive adhesions, inflammatory phlegmon at the head of pancreas, edematous cystic duct and hepatoduodenal ligament, presence of ascites, pseudocyst pancreas in retrogastric position.

NEWER APPROACHES IN LAPAROSCOPIC CHOLECYSTECTOMY

a) GASLESS LAPAROSCOPIC CHOLECYSTECTOMY:

Gasless LC is especially useful in patients with cardiorespiratory problems. Here the abdominal wall is lifted mechanically allowing an adequate space for laparoscopic surgery.

b) SPA (SINGLE PORT ACCESS) CHOLECYSTECTOMY.

METHODOLOGY

The materials for the present study on "A CLINICAL STUDY TO DETERMINE PREDICTIVE FACTORS FOR DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY" comprises of 50 cases admitted to Govt rajaji Hospital from March 2013 to August 2014 a period of 18 months.

The method for the study included screening of patients who presented with upper abdominal pain, or vomiting or dyspepsia or jaundice. Such patients were studied in detail clinically and investigated as per the proforma detailed below. Ultrasound abdomen was done in all patients.

Routine haematological and biochemical investigations were done. Investigations like OCG, PTC, PT-INR could not be done routinely due to lack of facilities. LFT was done in all patients. ERCP done in indicated patients, The patients confirmed by USG examination were evaluated with following factors: age, sex, h/o previous

hospitalization, BMI wt (kg)/ ht (mt^2), abdominal scar- supraumbilical or infraumbilical, palpable gall bladder, sonographic findings- wall thickness, Pericholecystic collection, impacted stone.

All the patients were received symptomatic treatment and vitamin K for 3 days preoperatively.

Following evaluation the patient will be subjected to laparoscopic cholecystectomy and time taken, biliary / stone spillage, injury to duct/ artery or conversion were noted. All the patients were operated by one surgical unit. Post operatively cases were followed up for any complication. Drain was removed between 2^{nd} and 5^{th} post OP day depending on the drainage, and Suture removal was done 8^{th} post OP day. All cases were followed up for any recurrent symptoms.

INCLUSION CRITERIA:

The patients aged between 16 and 60 yrs presenting with symptoms and signs of Cholelithiasis / Cholecystitis and diagnosed by USG examination in surgical ward of GRH - Madurai

EXCLUSION CRITERIA:

Patients below 15 years of age.

Patients with CBD calculus, raised ALP, dilated CBD, where CBD exploration was needed.

Patients with features of obstructive jaundice.

Patients refusing surgery.

Patients not willing for laparoscopic cholecystectomy.

RESULTS

This study included 50 cases that were studied prospectively over a period of 18 months, from March 2013 to August 2014

AGE DISTRIBUTION

In the present series the youngest patient was 19 yrs of age and the oldest was 60 yrs of age. Majority of the patients in the present series were in the age group of 31-40 yrs of age.

TABLE 10: Showing the age wise distribution of cholelithiasis.

AGE IN YEARS	PRESENT SERIES	%
0-10 yrs	0	0
11-20 yrs	1	2%
21-30 yrs	12	24%
31-40 yrs	15	30%
41-50 yrs	13	26%
51-60 yrs	9	18%
>61 yrs	0	0

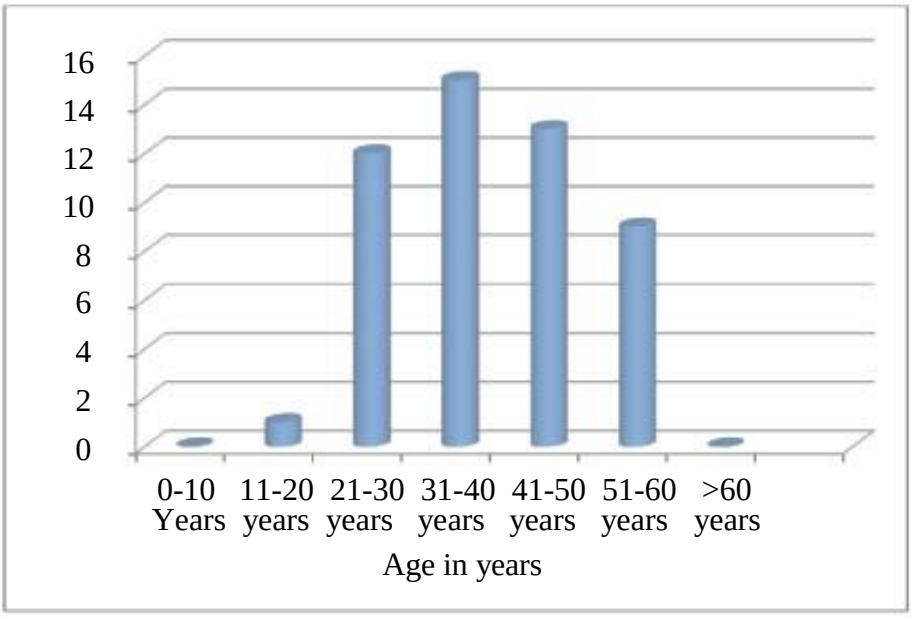


FIGURE 17: Graph showing age wise distribution of cholelithiasis.

SEX DISTRIBUTION

Out of 50 patients 35 were females and 15 were male patients. The male:female ratio is 1:2.3.

TABLE 11: Showing sex wise distribution of cholelithiasis

SEX	PRESENTSERIES	%
MALE	15	30%
FEMALE	35	70%

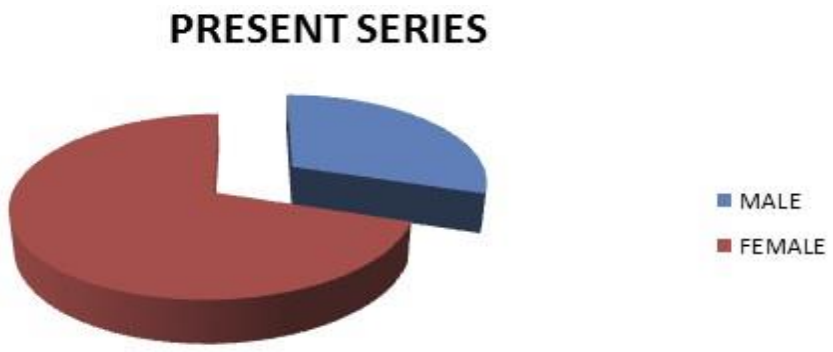


FIGURE 18: Pie diagram showing sex wise distribution of cholelithiasis

PRESENTING SYMPTOMS

Pain was the predominant symptom seen in all 50(100%) patients. Vomiting was present in 19 (38%) of the patients with pain. 1(2%) patient had jaundice and 11(22%) patients had dyspepsia.

TABLE 12: Showing presenting symptoms

SYMPTOMS	PRESENT SERIES	%
PAIN	50	100%
VOMITING	19	38%
JAUNDICE	1	2%
DYSPEPSIA	11	22%
FEVER	6	12%

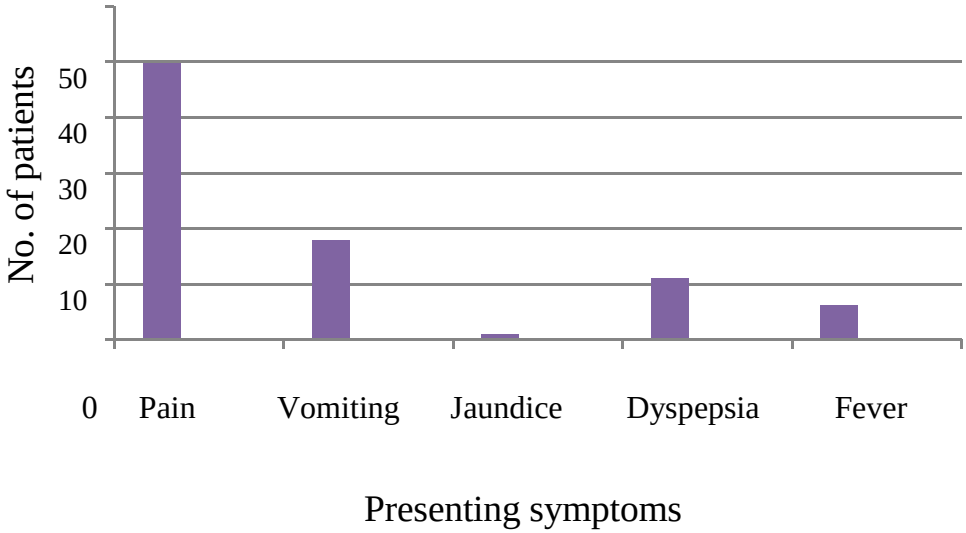


FIGURE 19: Graph showing presenting symptoms

PRESENTING SIGNS

Tenderness in right hypochodrium was present in 40(80%) patients, Guarding and rigidity in 2 (4%) patients and a mass was palpable in 4 (8%) patients.

TABLE 13: Showing presenting signs

SIGNS	PRESENT SERIES	%
TENDERNESS IN RIGHT HYPOCHONDRIUM	40	80
GUARDING	2	4
MASS	4	8

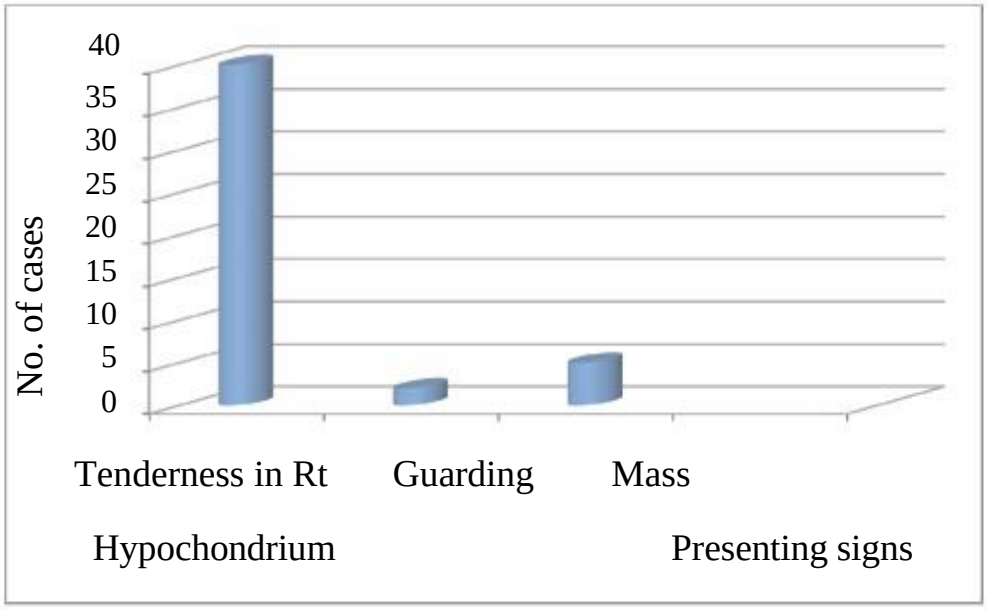


FIGURE 20: Graph showing presenting signs

CORRELATION WITH BLOOD GROUP

Of the 50 patients 23 patients had of blood group 'O', 15 patients had of blood group 'B', 10 patients had of blood group 'A' and 2 patients had blood group AB.

TABLE 14: Showing correlation with blood group

Blood Group	Present Series	%
A	10	20
B	15	30
AB	2	4
O	23	46

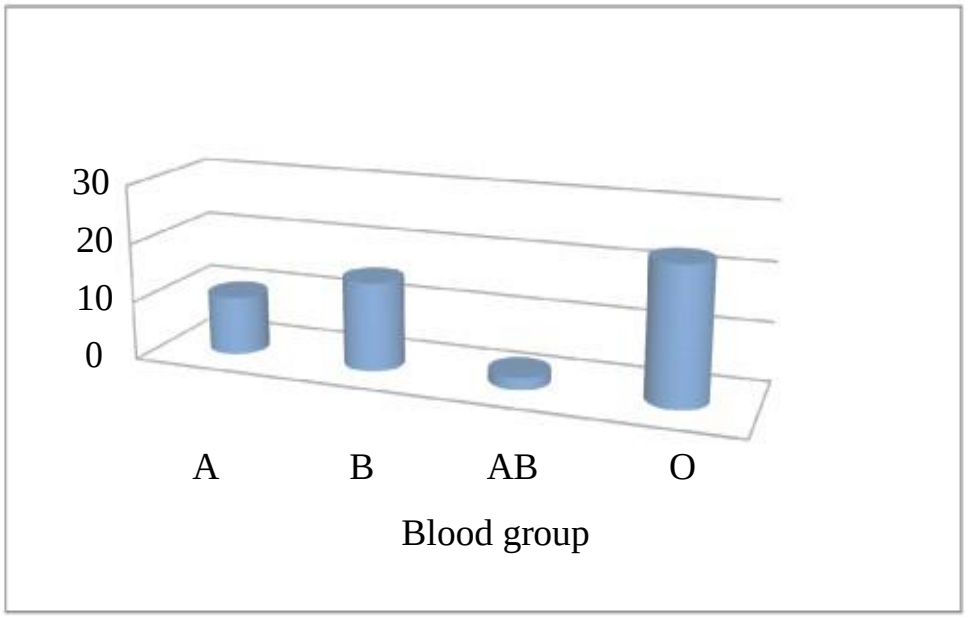


FIGURE 21: Graph showing correlation with blood group

ULTRASONOGRAPHY.

TABLE 15: Showing ultrasonography findings

ULTRASONOGRAPHY	NO OF CASES
Multiple calculi	33(66%)
Solitary calculi	9 (18%)
Solitary impacted calculi	8 (16%)
Wall thickening	19(38%)
Pericholecystic collection	8 (16%)

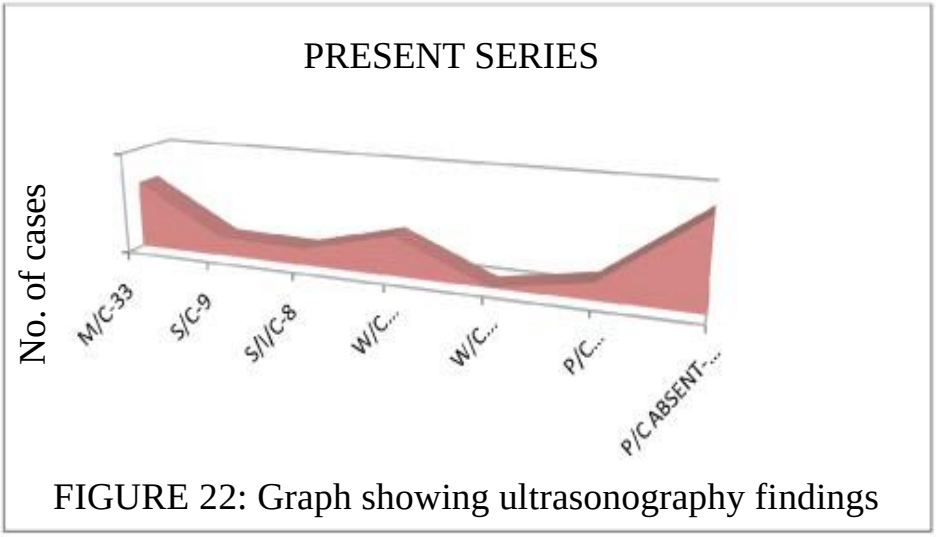


FIGURE 22: Graph showing ultrasonography findings

All the 50 patients had stones in gallbladder, 33 patients had multiple calculi, 9 had solitary calculi and 8 had solitary impacted calculi. , 19 patients had wall thickening and 8 had pericholecystic collection

CORRELATION OF PRE-OP SCORE AND THE OUTCOME

Out of the 5 patients in whom lap. was converted to open, 3 patients had Extensive adhesions with difficulty in dissection and 1 had uncontrolled bleeding from cystic artery which was anatomical variant. Another 1 had mass formation

TABLE 16: Showing correlation of pre-op score and the outcome

PRE-OP SCORE	EASY	DIFFICULT	VERY DIFFICULT	TOTAL
0-5	36	2	2	40
6-10	0	7	2	9
11-15	0	0	1	1
TOTAL	36	9	5	50

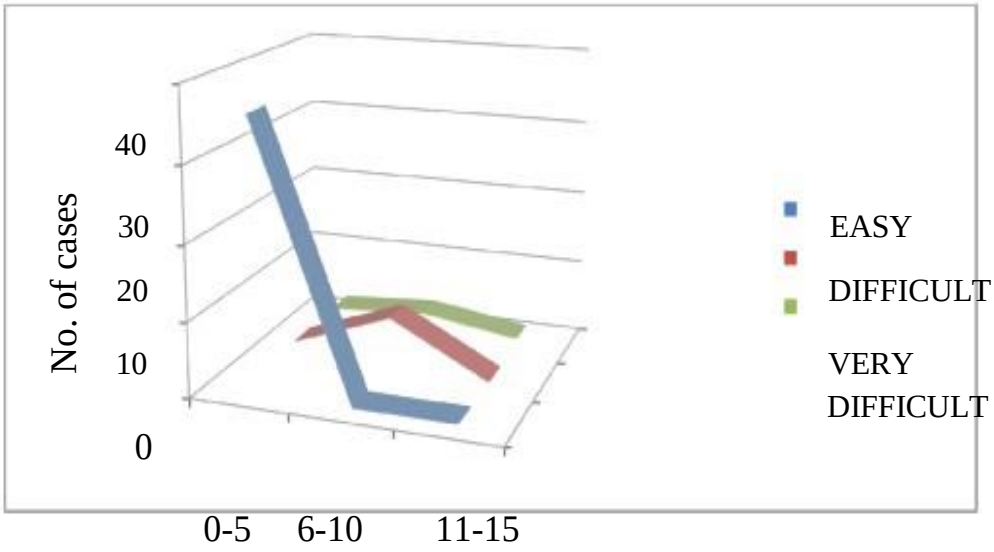


FIGURE 23: Graph showing correlation of preop score and the outcome

SCORING FACTORS AND EASY/DIFFICULT CRITERIA-ANNEXURE II/III

ANALYSIS OF PER-OPERATIVE OUTCOME WITH THE RISK FACTORS

TABLE 17:Showing the analysis of pre-operative outcome with the risk factors

RISK FACTORS	**LEVEL**	**PER-OP OUTCOME**		**P Valve**
		D-NO (%)	E-NO (%)	
AGE	<= 50 Y	10	29	1.000
	>50 Y	2	9	
SEX	FEMALE	9	25	0.6976
	MALE	3	12	
BMI wt(kg)/ht(m^2)	<=25	1	26	0.4324
	25.1-27.5	1	10	
	>27.5	10	2	<0.0001
PREVIOUS SURG.	Nil	6	27	0.6959
	yes	3	14	
HOSPITALIZATION	Nil	6	39	0.0008
	Yes	4	1	
GB PALPABLE	NP	9	39	0.0364
	Yes	2	0	
USG- WALL THICK	N	1	34	0.0001
	Yes	9	6	
IMPACTED STONE	Nil	7	37	0.0103
	Yes	4	2	
P/C COLLECTION	Nil	6	34	0.0471
	Yes	6	4	

$0.01 < p \leq 0.05$ moderately significant and $p \leq 0.01$ Highly significant if $p < 0.001$ strongly significant

D-Difficult E-Easy NP-Non palpable N-Normal P value-Predictive value. In the present study prior hospitalization, BMI >27.5, Palpable GB, Thick GB wall, Impacted stone and Pericholecystic collection were significant predictors of difficult laparoscopic cholecystectomy. Fischer exact test was used to find the significant association of findings of preoperative score with per operative outcome.

POST-OPERATIVE COMPLICATION

Only 2 patients had infection of the epigastric port site which required required cleaning and dressing daily.

TABLE 18: Showing postoperative complications

POST OPERATIVE COMPLICATION	NO. OF CASES
Wound infection	2
Haemorrhage	0
Retained stone	0
Bile leak	0
Prolonged ileus	0

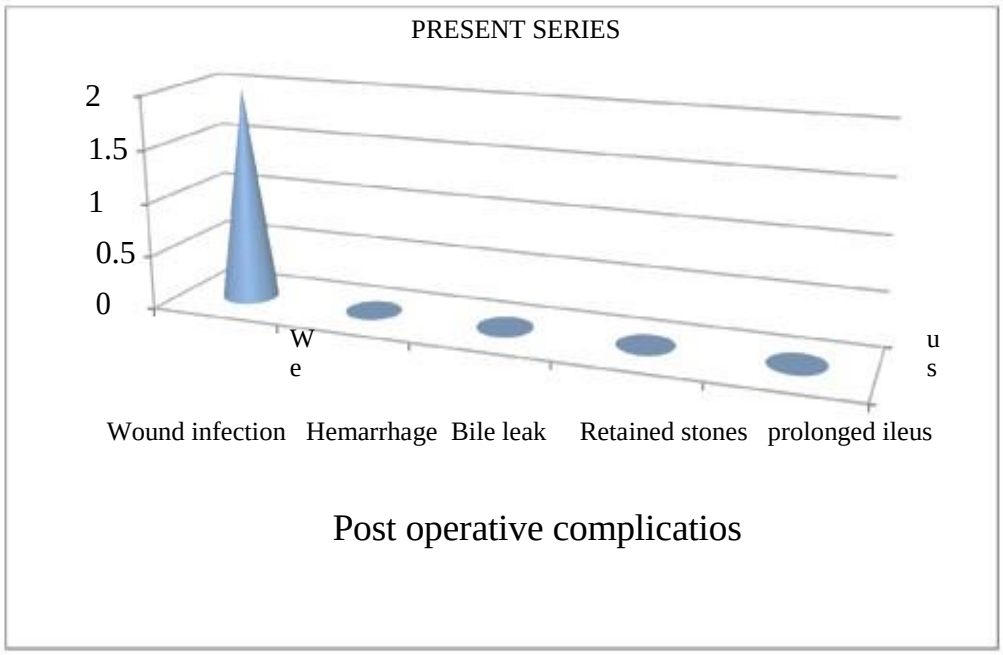
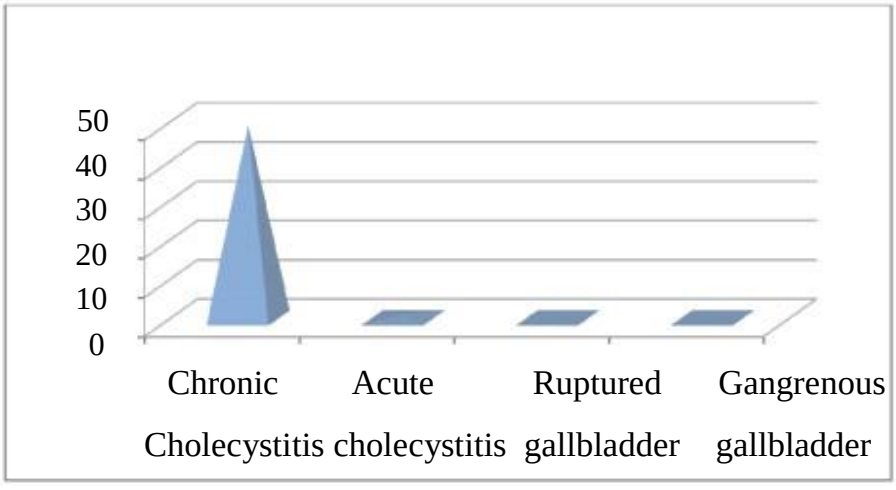


FIGURE 24: Graph showing post operative complication

HISTOPATHOLOGICAL EXAMINATION

49 cases were reported as chronic cholecystitis, while one was reported as acute cholecystitis. No case of malignancy of the GB was detected



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HISTOPATHOLOGIC EXAMINATION	NO. OF CASES
Chronic cholecystitis	49
Acute cholecystitis	1
Ruptured gall bladder	0
Gangrenous gall bladder	0

DISCUSSION

AGE DISTRIBUTION

In my study majority of the patients in the present series were in the age group of 31-40 yrs of age, and 80% 0f the patients came under the age group from 21-50 years

According to my study age is not a significant predictor since majority of the patients had easy cholecystectomy irrespective of age

SEX DISTRIBUTION

In the present series, out of 50 patients 35 were females and 15 were male patients. The male : female ratio is 1:2.3.

Endogenous estrogen and progestin are attributed to this phenomenon

Sex is not a significant predictor in my study

PRESENTING SYMPTOMS

PAIN

Pain was the predominant symptom seen in all 50 patients. All the 50 patients presented with chronic recurring pain. In 82% (41) of patients pain was in the right hypochondrium. Of the 41 patients, 72% (36) patients had colicky type of pain, 28%(14) patients had gripping type of pain and 18% (9) patients had dull aching type of pain. In 18% (9) patients, pain was in epigastrium predominantly. Radiation of pain to back was seen in 28%.

No asymptomatic patients in my study group

VOMITING

Vomiting was present in 38% (19) of the patients with pain. Vomiting was spontaneous and occurred mostly during the attack of pain. It indicates severity of disease in my study group

JAUNDICE

Jaundice was present in 1 patient, which was obstructive in nature associated with pain and fever, The patient underwent ERCP with CBD stenting. It was followed by cholecystectomy after 6 weeks which was easy as predicted.

It stated that 6 weeks duration required to settle inflammation significantly.

DYSPEPSIA

Dyspepsia was present in 22% (11) of the patients. On endoscopy 3 of them had duodenal ulceration.

Dyspepsia of another cause may coincide with cholelithiasis

FEVER

Fever was present in 12% (6) of the patients which was of moderate degree and was associated with chills.

In my study patients with fever invariably associated with increased gall bladder wall thickness and pericholecystic fluid collection those patients had difficult cholecystectomy, hence fever is a strong predictor of difficulty

PAST HISTORY

Of the 50 patients, 11 had undergone tubectomy, 2 had undergone LSCS, 1 had undergone appendicectomy, and 1 had undergone hysterectomy. 1 patient presented with obstructive jaundice due to CBD calculus, and he underwent ERCP with CBD stenting. 3 patients had attack of acute cholecystitis which required hospitalization and were managed conservatively. One patient had acute pancreatitis and was treated conservatively with hospitalization.

Previous surgeries didn't affect the per operative outcome significantly, since patients in my study underwent lower abdominal surgeries rather than upper abdominal surgeries

According to my study patients with history of previous hospitalisation had difficult cholecystectomy, hence it is a significant predictor of difficulty

PERSONAL HISTORY

Only 2 patients in the present series were purely vegetarian in their diet, while the remaining had mixed dietary habits. 9(60%) of the 15 male patients consumed alcohol regularly.

None of the female patients consumed alcohol.

It stated that Alcohol is a important risk factor

FAMILY HISTORY

None of the patients in the present series had a family history of cholelithiasis.

GENERAL PHYSICAL EXAMINATION

General survey revealed that 27(54%) patients had BMI < 25, 11 (22%) had BMI in the range of 25-27.5, and 12 (24%) had BMI > 27.5.

Among 12 patients 4 patients were hypertensive and 2 were diabetic. 1 patient had LRI . 1 patient was a known case of hypothyroidism and was on thyroid hormone supplementation.

On inspection, scar due to previous surgery was seen in 17(34%) of the patients. Out of this all were infraumbilical scar.

According to my study Obese patients had difficult cholecystectomy and BMI is a strong predictor and obesity associated with other co morbid conditions like diabetes and hypertension

PRESENTING SIGNS

Tenderness in right hypochondrium was present in 40(80%) patients. guarding and rigidity was present in 2(4%) patients Murphy's sign was present in 11(22%) patients. Mass was palpable in 2(4%) patients

Guarding and rigidity with mass was a sign of acute inflammation, associated with ultrasonagram findings favour for difficult cholecystectomy.

INVESTIGATION

Routine biochemical and hematological investigations like Hb%, Urine examination, Blood grouping, B.urea, S.ceatinine, RBS and LFT were done in all cases.

Hb% of patients ranged from 10 to 13 gm%. FBS and PPBS were done for diabetic patients. B.urea and S.creatinine were within normal limits.

One patient had deranged LFT with raised SGOT and SGPT levels. Majority of patients in present series belonged to Blood group 'O' constituting about 46%. 30% and 20% had blood group 'B' and 'A' repectively, Only 4% had blood group 'AB'.

Patient with deranged LFT had CBD stone, that patient sudjected to ERCP and after 6 weeks interval cholecystectomy done which was easy

It stated that Gall stone disease with deranged LFT need further investigation and delayed cholecystectomy also influences the per operative outcome

ULTRASONOGRAPHY

Ultrasound was done as a routine investigation in all the patients. The sonologic criteria used to diagnose gall stones were acoustic shadowing of the opacities in the gall bladder and change in the position of the opacity with the change in patient position.

All the 50 patients had stones in gallbladder, 15 patients had wall thickening and 10 had pericholecystic fluid collection.

33 patients had multiple calculi, 9 had solitary calculus and 8 had solitary impacted calculi.

As per my study GB wall thickness and pericholecystic fluid collection are strong predictors of difficulty

EVALUATION OF PREDICTIVE FACTORS FOR DIFFICULT LAPAROSCOPIC CHOLECYSTECTOMY:

The factors included were age, sex, prior H/O hospitalization for acute cholecystitis/ biliary pancreatitis/ obstructive jaundice due to CBD calculus, BMI, abdominal scar due to previous surgery, clinically palpable GB, wall thickness, pericholecystic fluid collection, impacted stone.

CORRELATION OF PRE-OP SCORE AND THE OUTCOME

Out of the 5 patients lap converted to open in 5 patients since 3 had extensive adhesions, 1 had aberrent anatomy another 1 had mass formation

The positive predictive value for easy prediction was 94.7% and for difficult prediction was 100%.

Conversion rate from lap. cholecystectomy to open cholecystectomy was 10% in the present series.

POST-OPERATIVE TREATMENT

a) Nasogastric aspiration till the patient recovered from the postoperative ileus evidenced from appearance of bowel sounds and passage of flatus.

b) I-V fluids continued till oral liquid diet was started, ie following removal of Ryle's tube.

c) Broad spectrum antibiotic for 5 days

d) Analgesics as and when required

e) Drainage tube was removed between 1^{st} and 5^{th} post OP day.

POST-OPERATIVE COMPLICATION

Only 2 patients had infection of the epigastric port site which required clean and dressing daily. It healed by secondary intention.

As per my study all patients received better post operative care, Since Difficulties predicted already major complications avoided

HISTOPATHOLOGICAL EXAMINATION

49 cases were reported as chronic cholecystitis (includes acute on chronic), while one was reported as acute cholecystitis. No case of malignancy of the GB was detected

FOLLOW UP

All patients were followed up for a period of 1 month and no significant complication was noted.

CONCLUSIONS

Accoring to my study

1.Age and sex of the patients are not a significant predictors

2.The incidence of gall stones was found to be more in patients with blood group O

3.Pain was the predominant symptom seen in all (100%) the patients.

4.BMI ($p<0.001$)is a strong significant predictor, obesity associated with other co morbid conditions also

5.Previous history of hospitalisation ($p<0.0008$) for acute cholecystitis, acute pancreatitis, and obstructive jaundice are significant predictors of difficult lap cholecystectomy.

6.Previous surgeries not a significant predictors

7.Alcohol is a important risk factor in gall stone disease

8.Palpable GB ($p<0.0364$) is a significant predictor

9.USG findings ($p<0.0471$) are strong predictors of difficult surgery.

10.The conversion rate from laparoscopic cholecystectomy to open cholecystectomy was 10%

11.The incidence of port site infections was 4%

12.Histopathological examination revealed chronic cholecystitis in 98% of cases and acute cholecystitis in 2%.

In the present study, BMI >27.5 ($P<0.001$), history of prior hospitalization ($P<0.0008$), palpable gallbladder ($p<0.0364$), impacted stone ($P<0.0103$) and Pericholecystic fluid collection ($P<0.0471$) were significant predictors of difficult laparoscopic cholecystectomy. positive predictive value for easy prediction was 94.7% and for difficult prediction was 100%.

SUMMARY

Cholelithiasis is the most common biliary pathology. Gall stone are present in10 to 15% of the general population and asymptomatic in the majority of them, of about >80%. Approximately 1-2% of asymptomatic patients will develop symptoms requiring cholecystectomy every year, making it one of the most common operations performed.

In 1992, The National Institute of Health (NIH) consensus development Conference stated that laparoscopic cholecystectomy "Provides a safe and effective treatment for most patients with symptomatic gallstones".

In about 5 to 10% of the cases of laparoscopic cholecystectomy, conversion to open cholecystectomy may be needed for safe removal of gallbladder.

Therefore it is necessary to analyse the risk factors that predict difficult laparoscopic cholecystectomy.

The following risk factors were considered- age>50 years, male sex, H/O prior hospitalization for acute cholecystitis/ biliary

pancreatitis, BMI 25-27.5 and >27.5, abdominal scar, palpable GB, wall thickening, impacted stone, and pericholecystic collection. Out of this BMI >27.5, H/O prior hospitalization for acute cholecystitis/acute pancreatitis, palpable GB, wall thickening, impacted stone, and pericholecystic fluid collection were significant predictors of difficult laparoscopic cholecystectomy, as per present study.

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ANNEXURE-I

PROFORMA

NAME	IP NO
AGE	DOA
SEX	DOO
RELIGION	DOD
OCCUPATION	
ADDRESS	

I. PRESENTING COMPLAINTS

A. PAIN

B. FLATULENT DYSPEPSIA

C. NAUSEA AND VOMITING

D. JAUNDICE

E. APPETITE

F. FEVER

G. MASS PER ABDOMEN

H. BOWEL HABITS

II. HISTORY OF PRESENTING ILLNESS

a. PAIN

Site

Duration

Character

Radiation

Relation to Food

Aggravating and relieving factors

b. FLATULENT DYSPEPSIA

Epigastric discomfort

Belching

Heart burn

c. NAUSEA AND VOMITING

Frequency

Character (whether bilious or not)

Relief after vomiting

Relationship to food

d. JAUNDICE

Mode of onset (gradual or sudden) Intermitent or persistent

Duration

Progression/Painless Or Painful/Depth

High coloured urine

Pruritis

e. APPETITE

Dislike for fatty foods

f. FEVER

Intermitent with rigors

g. MASS PER ABDOMEN

Site

Duration

Association with pain

h.BOWEL HABITS

Colour of stools (white or clay coloured stools)

Constipation

III. PAST HISTORY

H/O similar complaints in the past

H/O of acute cholecystitis and previous hospitalisation

H/O jaundice

H/O ocp ingestion

H/O previous surgery or ERCP

IV.PERSONAL HISTORY

Appetite

Sleep

Diet

Bowel/Bladder

Habits

Menstrual H/O

No of children

V.FAMILY HISTORY

H/O Any family members suffering from similar complaints

Family H/O DM, HTN

VI. GENERAL PHYSICAL EXAMINATION

BMI

Pulse BP

Temperature

Pallor

Icterus

Cyanosis

Clubbing

Koilonychia

Lymphadenopathy

Pedal Edema

VII. PER-ABDOMEN EXAMINATION

INSPECTION

Contour	
Movement with respiration	
Skin	Umbilicus
Visible swelling	Site
	Size
	Shape
	Borders
	Surface
Visible peristalsis	
Operative scars/ Sinuses/ Dilated veins/ Visible pulsations	
Hernial orifices	
External genitalia	

PALPATION

Tenderness	
Murphy's sign	
Boa's sign	
Palpable mass	Present/ Absent: if present
	Tenderness
	Local rise of temperature

Site

Surface

Mobility

Border

Plane of the swelling

Consistency

Movement with respiration

Other masses

PERCUSSION

Liver dullness and span

Percussion note over the mass (if present)

Shifting dullness

AUSCULTATION

Bowel sounds

BACK

PV/PR

VII.SYSTEMIC EXAMINATION

Cardiovascular system

Respiratory system

Nervous system

INVESTIGATIONS

Hb%

BT, CT

Total count and Differential count

ESR

Urine Albumin

Sugar

Microscopy

Bile salts/ Bile pigments

FBS

B. Urea

S. Creatinine

Blood group

PT-INR

LFT

Total bilirubin

Direct bilirubin

SGOT

SGPT

Albumin

Alkaline phosphatase

ECG

Ultrasound abdomen

Stone or Sludge

Impacted stone

Post ERCP status

Wall thickness

Pericholecystic collection

CBD & intrahepatic biliary radicals

Portal vein

Liver

DIAGNOSIS

OPERATIVE DETAILS

Anaesthesia

Time taken

Bile/stone spillage

Injury to duct/artery

Conversion to open

Reason for conversion

POST-OPERATIVE PERIOD

Drain removal

Suture removal

Wound infection/ hemarrhage / Bile leak / Prolonged ileus / Retained stone

FOLLOW UP

All patients were followed up for a period of one month.

ANNEXURE-II

SCORING FACTORS

HISTORY			**MAX. SCORE**
AGE	<50y(0)	>50y(1)	1
SEX	Female(0)	Male(1)	1
H/O HOSPITALIZATION	N(0)	Y(4)	4
CLINICAL			
BMI wt(kg)/ht(m^2)	<25 (0)	25-27.5(1) >27.5 (2)	2
ABDOMINAL SCAR	N (0)	Infra-imbilical(1) Supra-umbilical(2)	1
PALPABLE GB	N (0)	Y (1)	1
SONOGRAPHY			
WALL THICKNESS	Thin (0)	Thick >4mm(2)	2
PERICHOLECYSTIC COLLECTION	N (0)	Y (1)	1
IMPACTED STONE	N (0)	Y (1)	1

TOTAL MAXIMUM SCORE - 15

N – NO, Y – YES, H/O - HISTORY OF.

ANNEXURE-III

EASY/DIFFICULT CRITERIA

EASY	Time taken <60 min No bile spillage No injury to duct, artery
DIFFICULLT	Time taken 60-120 min Bile/stone spillage Injury to duct No conversion
VERY DIFFICULT	Time taken >120 min Conversion

MASTER CHART

Sl.No	Name	IP No	Age(yrs)	Sex	Pain				Vomiting	Fever	Dyspepsi	Past History			Personal History			GPE	P/A	Palpation			Hb%	Blood group	LFT	USG			Lap Cholecystectomy details				Conversion to open	Drain removal	Postop Pd	Predictive score	Lap Chole Eat
					Duration	Location	Character	Radiation				Jaundice	Surgery	Comorbidities	Alcohol	Diet	BMI		Inspection	Tenderness	Mass	Murphy's sign				No of calculi	GB wall thick	Pericholecol	Adhesions	Time taken	S/S Spillage	Injuries					
1	Ponrai	11069	38	M	6m	RHC	Colicky	Back	+	-	-	-	-	-	+	Mi		20.16	NAD	RHC	-	-	11.6	O+ve	N	M/C	-	-	-	45mins	-	-	-	POD2	NS	1	E
2	Maheswari	17302	27	F	1yr	RHC	Colicky	Back	-	-	-	-	Tubectomy	-	-	Mi		17.48	Scar+	RHC	-	-	10.2	B+ve	N	M/C	+	-	+	50min	-	-	-	POD2	NS	3	E
3	Pitchai	18006	47	M	4yr	Epg	Dull	-	+	-	+	-	-	Ac. Pancreatitis	+	Mi		24.81	NAD	RHC	-	-	11.2	A+ve	N	M/C	+	-	+	1hr05min	-	-	-	POD4	NS	7	D
4	dhanalakshmi	20969	28	F	1yr	RHc	Colicky	-	+	-	-	-	Tubectomy	-	-	V		22.18	Scar+	RHC	-	+	10.6	O+ve	N	S/I/C	+	+	+	55min	-	-	-	POD4	NS	5	E
5	Ganesan	21065	57	M	6m	RHC	Colicky	-	+	-	-	-	-	DM & HTN	+	Mi		25.16	NAD	RHC	-	+	11	A+ve	N	M/C	+	-	+	50min	+	-	-	POD3	NS	5	E
6	Arthi	22725	21	F	8m	RHC	Colicky	Back	-	+	-	-	Acute Cho.	-	-	Mi		34.24	NAD	RHC	+	-	11.6	A+ve	N	S/I/C	+	-	+	-	-	+	+	POD3	NS	10	VD
																												Aberrent anatomy								
7	Sasikala	22988	34	F	6m	RHC	Gripping	-	-	-	-	-	-	-	-	Mi		19.53	NAD	RHC	-	-	10.8	O+ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	0	E
8	Menaka	23347	40	F	5m	Epg	Dull	-	+	-	+	-	Tubectomy	-	-	Mi		23.9	Scar +	Epg	-	-	10	B+ve	N	M/C	-	-	+	50 min	-	-	-	POD3	NS	1	E
9	Padma	27349	48	F	6m	RHC	Colicky	Back	-	-	-	-	-	HTN	-	Mi		22.49	NAD	RHC	-	+	10.8	A+ve	N	S/C	-	-	+	55min	-	-	-	POD4	NS	0	E
10	Suganthi	28303	39	F	8m	RHC	Colicky	Back	+	+	-	+	ERCP	GDM+	-	Mi		28.9	NAD	-	-	-	11	B+ve	N	S/C	+	-	+	1hr15min	+	-	-	POD3	NS	8	D
11	Marisamy	29517	40	M	5m	RHC	Colicky	-	-	-	-	-	-	-	+	Mi		20.06	NAD	RHC	-	-	12.8	O+ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	1	E
12	Thangarai	29550	57	M	3m	RHC	Colicky	-	-	-	-	-	Appen.	-	-	Mi		25.39	Scar+	RHC	-	-	12	B+ve	N	M/C	-	-	+	55min	-	-	-	POD2	NS	5	E
13	Pandi	31849	27	M	5m	RHC	Colicky	-	-	-	-	-	-	-	-	Mi		19.53	NAD	RHC	-	-	11.6	B+ve	N	M/C	-	-	-	45mins	-	-	-	POD2	NS	1	E
14	Ayeesha	35145	40	F	6m	Epg	Dull	-	+	-	+	-	-	Br.Asthma	-	V		28.06	NAD	Epg	-	-	11.1	O+ve	N	S/I/C	+	+	+	1hr15min	+	-	-	POD3	W/I +	10	D
														Acute cho.																							
15	Shobha	38707	40	F	8m	RHC	Colicky	-	-	-	-	+	-	Splenec.	HTN	-	Mi		25	Scar+	RHC	-	-	9.6	A+ve	N	M/C	-	-	-	55min	-	-	-	POD3	NS	3	E
16	Heena Banu	41109	19	F	9m	RHC	Colicky	Back	-	-	-	-	-	-	-	Mi		17.94	NAD	RHC	-	-	10.6	B+ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	0	E
17	Nagarajan	41930	24	M	6m	RHC	Colicky	-	+	-	-	-	-	-	+	Mi		24.22	NAD	RHC	-	+	11.8	O+ve	N	S/C	-	-	+	55min	-	-	-	POD2	NS	1	E
18	Paniroja	43870	60	F	6m	RHC	Colicky	-	-	-	-	-	-	-	-	Mi		20.93	NAD	RHC	-	-	10.6	O+ve	N	M/C	+	+	+	55min	-	-	-	POD3	NS	5	E
19	Indira	43526	41	F	1yr	Epg	Dull	-	-	-	+	-	Tubectomy	-	-	Mi		20.44	Scar+	Epg	-	-	10	B+ve	N	S/C	-	-	-	40min	-	-	-	POD3	NS	1	E
20	Rajammal	48151	28	F	7m	RHC	Gripping	-	+	+	-	-	-	-	-	Mi		25.77	NAD	RHC	-	+	11	A+ve	N	M/C	+	-	+	50min	+	-	-	POD4	NS	2	E
21	Kalyani	49267	30	F	6m	RHC	Colicky	-	-	-	-	-	-	-	-	Mi		25.39	NAD	RHC	-	-	11	O+ve	N	M/C	-	-	-	45min	-	-	-	POD2	NS	1	E
22	Nazrin Begum	51211	38	F	6m	RHC	Colicky	-	-	-	-	-	Tubectomy	-	-	Mi		20.54	Scar+	RHC	-	-	10.2	O-ve	N	S/I/C	-	-	-	40min	-	-	-	POD1	NS	2	E
23	Lakhmi Devi	55764	45	F	8m	Epg	Dull	-	-	-	+	-	LSCS	Hypothyroidism	-	Mi		27.91	Scar+	Epg	-	-	10	O+ve	N	M/C	-	-	+	1hr05min	-	-	-	POD2	NS	3	D
24	Nagammal	56861	26	F	6m	RHC	Colicky	-	-	-	-	-	-	-	-	Mi		22.66	NAD	RHC	-	-	11	A+ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	1	E
25	Prema.M.	57773	51	F	5m	RHC	Colicky	-	-	-	-	-	-	-	-	Mi		23.82	NAD	RHC	-	-	10.5	O+ve	N	S/C	-	-	-	45min	-	-	-	POD2	NS	1	E
26	Kamala	58515	58	F	6m	RHC	Colicky	Back	+	-	-	-	Hysterect.	RFA for SVT	-	Mi		28.06	Scar+	RHC	-	+	11	B+ve	N	M/C	+	-	-	1hr	+	-	-	POD3	NS	6	D
27	Usha Rani	59569	23	F	1y	RHC	Colicky	-	-	-	-	-	-	Acute cho.	-	Mi		27	NAD	RHC	+	+	11	O-ve	N	S/I/C	+	+	+	1hr05min	+	-	-	POD3	NS	10	D
28	Sonaiammal	63849	25	F	6m	RHC	Colicky	-	-	-	-	-	Tubectomy	-	-	Mi		20.88	Scar+	RHC	-	-	10	B-ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	1	E
29	Rani	66886	37	F	5m	RHC	Gripping	-	-	-	-	-	Tubectomy	-	-	Mi		27.34	Scar+	RHC	-	-	11	O+ve	N	S/C	-	-	-	55min	-	-	-	POD2	NS	2	E
30	Ochadevar	68975	35	M	6m	RHC	Colicky	Back	-	-	-	-	-	-	-	Mi		23.12	NAD	RHC	-	-	12	O+ve	N	M/C	-	-	-	45min	-	-	-	POD2	NS	1	E
31	Kavitha	70046	25	F	5m	Epg	Dull	-	-	-	+	-	LSCS	-	-	Mi		20.44	Scar+	Epg	-	-	11	B+ve	N	M/C	-	-	-	55min	-	-	-	POD2	NS	1	E
32	Nataraian	73870	43	M	8m	RHC	Colicky	-	-	-	-	-	-	-	+	Mi		27.34	NAD	RHC	-	-	12	A+ve	N	M/C	-	-	-	50min	-	-	-	POD2	NS	2	E
33	Saritha	76073	43	F	6m	RHC	Colicky	Back	+	-	-	-	-	-	-	Mi		30.61	NAD	RHC	-	+	10	O-ve	N	S/I/C	+	+	+	1hr	+	-	-	POD3	NS	6	D
34	Janaki	75385	40	F	6m	RHC	Colicky	-	-	-	-	-	Tubectomy	-	-	Mi		24	Scar+	RHC	-	-	10	O+ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	1	E
35	Umavathy	74674	42	F	5m	RHC	Colicky	Back	+	+	-	-	-	-	-	Mi		28.88	NAD	RHC	-	+	10.6	AB+ve	N	M/C	+	-	+	1hr10min	+	-	-	POD4	W/I +	4	D
36	Saroja	76998	60	F	2y	Epg	Dull	-	-	-	+	-	-	DM & HTN	-	Mi		28.57	NAD	Epg	-	-	11	O+ve	N	M/C	-	-	-	55min	-	-	-	POD2	NS	3	E
37	Ayyayu	78986	44	M	6m	RHC	Gripping	-	+	-	-	-	-	-	+	Mi		27.625	NAD	RHC	+	+	12	B-ve	N	S/I/C	+	-	-	1hr	+	-	-	POD2	NS	6	D
38	Muthukarupu	81243	48	M	4m	RHC	Colicky	-	+	-	-	-	-	-	-	Mi		24.16	NAD	RHC	-	-	12	O+ve	N	M/C	+	-	+	55min	-	-	-	POD2	NS	3	E

39	Rajapandi	82894	37	M	5m	RHC	Colicky	-	-	-	-	-	-	-	+	Mi	22.47	NAD	RHC	-	-	11	O-ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	1	E
40	Jayalakshmi	82625	38	F	6m	Epg	Dull	-	-	-	+	-	-	-	-	Mi	23.45	NAD	Epg	-	-	11	O+ve	N	S/C	-	-	-	45min	-	-	-	POD2	NS	0	E
41	Pushpa	84650	55	F	1yr	RHC	Colicky	Back	-	-	-	-	-	-	-	Mi	22.6	NAD	RHC	-	-	10	A+ve	N	M/C	-	-	-	40min	-	-	-	POD2	NS	0	E
42	Rajamani	85191	35	F	6m	RHC	Colicky	-	-	-	-	-	Tubectomy	-	-	Mi	25.42	Scar+	RHC	-	-	10	O+ve	N	M/C	-	-	-	45min	-	-	-	POD2	NS	2	E
43	Karthikumar	84576	28	M	1yr	RHC	Colicky	Back	+	-	+	-	-	-	-	Mi	24.08	NAD	RHC	-	+	11	B-ve	N	S/C	-	-	+	55min	+	-	-	POD3	NS	1	E
44	Parimalam	85876	48	F	6m	RHC	Colicky	-	+	+	-	-	-	-	-	Mi	23.55	NAD	RHC	+	+	11	O+ve	N	S/I/C	+	+	+	-	-	-	+	POD4	NS	4	VD
																												Adhesion							
45	Jayalakhmi	88934	30	F	5m	RHC	Grippin g	-	-	-	-	-	-	-	-	Mi	23.06	NAD	RHC	-	-	10.5	B-ve	N	M/C	-	-	-	45min	-	-	-	POD2	NS	0	E
46	Kalyani	92314	45	F	6m	Epg	Dull	-	-	-	+	-	Tubectomy	-	-	Mi	24.75	Scar+	Epg	-	-	11	A+ve	N	M/C	-	-	-	50min	-	-	-	POD2	NS	1	E
47	Parvathy	94367	35	F	1yr	RHC	Colicky	Back	+	+	-	-	Tubectomy	-	-	Mi	30.08	Scar+	RHC	-	+	11	B-ve	N	M/C	+	+	+	-	-	-	+	POD4	NS	10	VD
													Acute Cho.														adhesion							
48	Fathiama	98760	49	F	7m	RHC	Colicky	-	+	-	-	-	-	-	-	Mi	28.88	NAD	RHC	+	+	10.6	O+ve	N	S/C	+	-	+	-	-	-	+	POD5	NS	5	VD
																												adhesion							
49	Veeran	97609	55	M	1yr	RHC	Colicky	Back	+	-	-	-	-	-	-	+	Mi	28.71	NAD	RHC	-	+	12	AB+ve	SGOT-112	M/C	+	+	Mass	-	-	-	+	POD4	NS	7	VD
																								SGPT-135											
																								TP-7.3											
																								Alb-3.1											
																								BiT-1.1											
																								BiD-0.4											
																								AIP-55											
50	Palaniyandi	0423	55	M	6m	RHC	Colicky	-	-	-	-	-	-	-	-	Mi	24.15	NAD	RHC	-	-	10.4	O+ve	N	M/C	-	-	-	50min	-	-	-	POD2	NS	2	E

KEY TO MASTER CHART

Alb	-Albumin
ALP	-Alkaline Phosphatase
Age(yrs)	-Age in Years
Appen.	-Appendicectomy
Acute Cho.	-Acute cholecystitis
Br.Asthama	-Bronchial Asthama
BMI	-Body Mass Index
BiT	-Total Bilirubin
BiD	-Direct Bilirubin
B/S Spillage	-Bile/Stone Spillage
CBD	-Common Bile Duct
D	-Difficult Category
DM	-Diabetes Mellitus
E	-Easy Category
Epg	-Epigastrium
ERCP	-Endoscopic Retrograde Cholangiopancreatography
F	-Female
GB	-Gall Bladder
GDM	-Gestational Diabetes Mellitus
GPE	-General Physical Examination
Hysterect.	-Hysterectomy
Hb	-Haemoglobin
HTN	-Hypertension

Lap.	-Laparoscopy
LFT	-Liver Function Test
LSCS	-Lower Segment Caesarean Section
M	-Male
M/C	-Multiple Calculi
Mi	-Mixed diet
NS	-Nothing Significant
N	-Normal
P/A	-Per Abdomen
PT-INR	-Prothrombin International Normalized Ratio
POD	-Post Operative Day
RHC	-Right Hypochondrium
S/C	-Solitary Calculus
S/I/C	-Solitary Impacted Calculus
SGOT	-Serum Glutamic Oxaloacetic Transaminase
SGPT	- Serum Glutamic Pyruvate Transaminase
Sl.No.	-Serial Number
Splenec.	-Splenectomy
TP	-Total Protein
V	-Vegetarian Diet
VD	-Very Difficult Category
W/I	-Wound Infection
+	-Present
-	-Absent